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EFFECT OF ORAL L-CARNITINE ADMINISTRATION ON HAEMATO-BIOCHEMICAL PARAMETERS OF CAMELS (*Camelus dromedarius*)

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ABSTRACT

Six mature healthy camels were divided into two equal groups, where camels of first group served as control and kept on basal diet without feed additives and camels of second group were kept on oral L-carnitine (3g/day) for three weeks. At the end of the experiment, body weight gained was calculated and whole blood was used for determination of haematological parameters and harvested sera were used for estimation of serum biochemistry. The present findings revealed that, the body weight gain in L-carnitine treated camel was comparable to the control untreated group. There were significant increase in the mean values of total leucocyte count (TLC) (18.1 ± 1.0 10^9/L) and neutrophils per cent (11.0 ± 1.0%) in L-carnitine treated camels as compared to that of control group where these values were 15.0 ± 0.5 10^9/L and 7.9 ± 0.1%, respectively. Biochemical analysis of serum revealed non-significant changes of all examined parameters. The examined oral dose of L-carnitine was immune-supportive and did not affect weight gain and biochemistry of dromedary camels. Unlike other animal species, this dose of L-carnitine did not affect lipid profile of dromedary camels.

Key words: Biochemistry, camels, haematology, L-carnitine, metabolism

L-carnitine plays an important role in mitochondrial β-oxidation of fatty acids as acyl carrier through the inner mitochondrial membrane in addition to its second function as a buffer for excess acyl residues (Murray *et al,* 2012). L-carnitine (3-hydroxy-4-N-trimethylaminobutirate) is a water-soluble quaternary amine that is synthesised mainly in liver from lysine and methionine of all mammals. About 80% of L-carnitine is present in muscle and about 5 to 10% in the gastrointestinal tract whereas the liver and blood contains only about 3% and 0.25% of the body's L-carnitine, respectively (Flores *et al,* 1996). L-carnitine is metabolically essential for the transport of long chain fatty acids from the cytosol into the mitochondrial matrix for β-oxidation. The activity of fatty acid β-oxidation enzyme, carnitine palmitoyl transferase (CPT) is increased significantly by L-carnitine supplementation (Arslan *et al,* 2003). Carnivores (cats and dogs) are unable to meet their L-carnitine requirement through endogenous synthesis in the long term since these animals have become adapted to an L-carnitine-rich diet in meat. As a consequence, the liver has probably lost its capacity for sufficient endogenous synthesis of L-carnitine (Harmeyer, 2002). In humans, 75% of the carnitine used by the body comes from the diet and the liver and the kidneys synthesise the remaining 25% from the immediate precursor gamma butyrobetain (Maritza *et al,* 2006). Dietary supplementation of L-carnitine reduces plasma very low-density lipoprotein cholesterol (VLDL-c) and triglycerides (TAG) levels in hyperlipidemic rabbits (Rajasekar *et al,* 2005). In another study on New Zealand rabbits, dietary L-carnitine reduced serum cholesterol, TAG, VLDL-c, low-density lipoprotein cholesterol (LDL-c) and increases glucose and high-density lipoprotein cholesterol (HDL-c) levels (Elgazzar *et al,* 2012). It has been postulated that, carnitine supplementation elevated urea concentration in growing steer (Greenwood *et al,* 2001). The effect of L-carnitine on lipid metabolism has been investigated in human (Delas *et al,* 2008), fish (Sang *et al,* 2012) and different animals (horse, cattle, sheep and pigs), birds (poultry, pigeon) and rodents (Harmeyer, 2003). However, the effect of L-carnitine in lipid metabolism is less known in camels. Therefore, the present study is aimed

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to investigate the effect of dietary L-carnitine on haematological and selected biochemical parameters in dromedary camels.

Materials and Methods

Location

A total of six mature healthy camels (450 ± 5 kg body weight; 4-7 years old) were kept in a wide, well ventilated and sunny stable with sand floor. These were fed approximately 3kg of barseem, 3kg cracked barley and 3kg concentrates in the morning and 3kg of barseem plus 3kg barley and 3 kg concentrates in the evening with free supply of mineral salt licks. Water was provided *ad libitum*. Camels were divided into 2 groups, three animals each. Camels of first group served as control and were kept on basal diet without feed additives. Camels of the second group were kept on L-carnitine tablets (Carniking^{TM}, Lonza, Fair Lown, NJ) at a dose of 3g/day given orally for three weeks. CarnikingTM (commercial product used in animal feed) contained 50% L-carnitine, 35% silica and 15% water.

At the end of the experiment, blood samples were collected from the jugular vein of all groups for estimation of total erythrocytic count (TEC), total leucocytic count (TLC), packed cell volume (PCV) and differential leucocytic count using standard haematological techniques (Drubkin, 1947; Feldman *et al,* 2000). Haemoglobin, Mean corpuscular volume (MCV), Mean corpuscular haemoglobin (MCH) and Mean corpuscular haemoglobin concentration (MCHC) were also estimated. Serum was separated by centrifu-gation of collected blood for 10 min at 1200g and was immediately frozen at –20ºC until the time of analysis. The sera were used for spectrophotometric determination of the activities of Aspartate Transaminase (AST) and Alanine Transaminase (ALT) as directed by Reitman & Frankle (1957). In addition, serum glucose, total protein, albumin and globulin values were determined spectrophotometrically as implied by the methods of Trinder (1969), Doumas *et al* (1981), Reinhold (1953) and Coles (1974), respectively. Serum blood urea nitrogen, uric acid and creatinine were determined according to the method described by Tabacco *et al* (1979) and Henry (1984), respectively. Furthermore, the obtained sera were used for spectrophotometric analysis of serum triacylglycerol (TAG) and total cholesterol by using of enzymatic method of spin react kits according to the methods of Sidney & Bernard (1973) and Zak *et al* (1954), respectively. Calcium, phosphorus and magnesium were determined by using commercial kits on chemistry analyser according to the manufacturer instructions.

Data were analysed by repeated measurements analysis of variance (ANOVA) and the statistical significance between means was compared using Student's t-test; $p<0.05$ was considered significant. All tests were performed using computer package of the statistical analysis system (SAS, 1987).

Results and Discussion

The data presented in Table 1 indicated that the body weight gain in L-carnitine treated camel was comparable to the control untreated group. Parallel to the current study, the addition of L-carnitine had no effect on average daily gain in ruminants (Greenwood *et al,* 2001). In addition, L-carnitine supplementation for 49 days exerted no effect on feed intake or live weight gain in merino lambs (Petek and Diniz, 2000). It must be taken into account that, under current experimental condition, the ruminal escape of the administered L-carnitine might have been low and the amount of L-carnitine available to camels might have been too small to affect performance parameters.

The results of haematological examination

Table 1. Effect of oral administration of L-carnitine (3g/day) for three weeks on body weight gain (Kg).

Groups	Body weight (Kg)		Gain in body weight (Kg)
	Initial	third weeks	
Control	506 ± 100	509 ± 100	3 ± 1
L-carnitine treated	546 ± 50	550 ± 40	3 ± 1

Values are mean ± SEM of 3 camels.

Table 2. Effect of oral administration of L-carnitine (3g/day) for three weeks on haematological parameters.

Variables	Control	L-carnitine treated
TEC (1012/L)	10.5 ± 2.0	10.6 ± 1.8
TLC (109/L)	15.0 ± 0.5	18.1 ± 1.0*
Neutrophils (%)	7.9 ± 0.1	11.0 ± 1.0*
Lymphocytes (%)	39.0 ± 5.1	31.5 ± 3.0
Esinophils (%)	4.5 ± 0.1	5.2 ± 1.0
Basophils (%)	0.7 ± 0.2	0.5 ± 0.2
Monocytes (%)	3.1 ± 0.2	3.6 ± 0.3
Hb (g/dl)	13.7 ± 1.5	13.8 ± 0.8
PCV (%)	27.6 ± 3.1	28.1 ± 1.0
MCV (fl)	27.0 ± 2.0	27.0 ± 2.0
MCH (pg)	13.2 ± 1.0	13.1 ± 1.0
MCHC (g/dl)	50.0 ± 2.0	49.1 ± 2.0

Values are mean ± SEM of 3 camels

*statistically significant when compared to control (group I) at $p< 0.05$

(Table 2) indicated that there were significant increase in the mean values of TLC in camels received 3g/day L-carnitine (18.1 ± 1.0 10^9/L) compare to the control group (15.0 ± 0.5). In addition, significant increase in the mean values of neutrophils was also observed in L-carnitine treated camels (11.0 ± 1.0%) compared to that of control group (7.9 ± 0.1%). Meanwhile, the mean values of TEC, lymphocytes, Eosinophil, Basophils, Monocytes, haemoglobin, PCV, MCV, HCV and MCHC showed non-significant variation in L-carnitine treated group (10.6 ± 1.8 10^{12}/L; 31.5 ± 3%; 5.2 ± 1%; 0.5 ± 0.2%; 3.6 ± 0.3%; 13.8 ± 0.8 g/dl; 28.1 ± 1.0 %; 27.0 ± 2.0 fl; 13.1 ± 1.0 pg; 49.1 ± 2.0 g/dl) compare to the control group (10.5 ± 2.0 10^{12}/L; 39.0 ± 5.1%; 4.5 ± 0.1%; 0.7 ± 0.2%; 3.1 ± 0.2%; 13.7 ± 1.5 g/dl; 27.6 ± 3.1 %; 27.0 ± 2.0 fl; 13.2 ± 1.0 pg; 50.0 ± 2.0 g/dl), respectively. The significant increase of TLC and neutrophil percentages in L-carnitine treated camels as compared to control was in accordance to the results of Famularo and De-Simone (1995) indicating the immune supportive effect of L-carnitine. In addition L-carnitine was postulated to enhance the phagocytic index in Nile tilapia (Abo-ghaneama *et al,* 2005). Moreover, L-carnitine is known to improve the neutrophil and macrophage function in aged rats at lower concentrations (Izgul-Uysal *et al,* 2004). Regarding the non-significant effect of L-carnitine on the rest of haematological parameters, similar findings (Rezaei *et al,* 2008) indicated that L-carnitine did not change the haematological parameters in laying hen fed 250mg of L-carnitine for every kg diet. Moreover, L-carnitine did not affect the haemoglobin content and TEC in broiler chickens (Asadi *et al,* 2013). Contrary by, it was found that L-carnitine caused leucopenia, decreased haemoglobin content and lowered the TEC with subsequent anemia in orally administered male albino rats (Qadir *et al,* 2008). Biochemical analysis (Table 3) indicated that glucose, total protein, albumin, globulin, A/G ratio, total cholesterol, triacylglycerol, ALT, AST, BUN, creatinine, calcium, phosphorus and magnesium showed non-significant variation in L-carnitine treated group (72 ± 3.0 mg/dl; 6.9 ± 1.12 g/l; 4.82 ± 0.1 g/l; 2.08 ± 0.3 g/l; 2.3 ± 0.2; 26.43 ± 1.5 mg/dl; 104.98 ± 1.7 mg/dl; 18.7 ± 1.07 U/l; 47.70 ± 2.0 U/l; 21.5 ± 1.2 mg/dl; 1.2 ± 0.1 mg/dl; 10.5 ± 0.03 mg/dl; 3.22 ± 0.70 mg/dl; 1.05 ± 0.01 mg/dl) compared to the control group (69.33 ± 1.0 mg/dl; 7.0 ± 1.11 g/l; 4.90 ± 0.1 g/l; 2.10 ± 0.2 g/l; 2.3 ± 0.39; 24.76 ± 1.3 mg/dl; 106.22 ± 1.1 mg/dl; 17.44 ± 1.13 U/l; 50.21 ± 0.76 U/l; 20.7 ± 1.34 mg/dl; 1.1 ± 0.2 mg/dl; 10.5 ± 0.05 mg/dl; 3.00 ± 0.60 mg/dl; 1.02 ± 0.00 mg/dl), respectively. Conversely to the current findings, previous reports (Arslan, 2003; Maritza *et al,* 2006; Elgazzar *et al,* 2012) reported a significant increase in TAG and total cholesterol levels on different animal species supplemented with L-carnitine. The reduction on TAG was postulated to removal of VLDL-c by lipoprotein lipase and beta oxidation (Tanaka *et al,* 2004) whereas, the reduction in cholesterol levels was attributed to conversion of cholesterol to bile acids (Seccombe *et al,* 1987). Conversely to the current study, it was documented that oral administration of L-carnitine reduced ALT and AST activities (Ahmed *et al,* 2010; Elgazzar *et al,* 2012; Sanjay and Singh, 2010). The results regarding the effect of L-carnitine on glucose levels are contradictory. Although previous reports (Greenwood *et al,* 2001; Elgazzar *et al,* 2012) indicated that, administration of oral L-carnitine induced an elevation of glucose levels on growing steer, Earlier work (Buyse *et al,* 2001) reported a reduction of glucose level in broiler chickens supplemented with L-carnitine. Currently, our findings indicated that oral administration of L-carnitine did not affect serum glucose level of dromedary camels. Earlier works (Greenwood *et al,* 2001; Salvatore *et al,* 2001; Elgazzar *et al,* 2012) described the nephrotoxic effect of L-carnitine on administered animals as reflected on their elevated levels of urea, creatinine, sodium potassium and chloride. However, the examined dose of the current study was safe to the kidney and liver function of dromedary camels. The present study concluded that the examined oral dose of L-carnitine was immune-supportive and did not affect weight gain and biochemistry of dromedary camels. Unlike

Table 3. Effect of oral administration of L-carnitine (3g/day) for three weeks on biochemical parameters.

Parameters	Control	L-carnitine treated
Glucose (mg/dl)	69.33 ± 1.00	72 ± 3.00
Total Protein (g/l)	7.00 ± 1.11	6.90 ± 1.12
Albumin (g/l)	4.90 ± 0.1	4.82 ± 0.1
Globulin (g/l)	2.10 ± 0.20	2.08 ± 0.30
A/G ratio	2.3 ± 0.39	2.3 ± 0.20
Total cholesterol (mg/dl)	24.76 ± 1.3	26.43 ± 1.5
TAG (mg/dl)	106.22 ± 1.1	104.98 ± 1.7
ALT (U/l)	17.44 ± 1.13	18.7 ± 1.07
AST (U/l)	50.21 ± 0.76	47.70 ± 2.00
BUN (mg/dl)	20.70 ± 1.34	21.5 ± 1.2
Creatinine (mg/dl)	1.10 ± 0.2	1.2 ± 0.1
Calcium (mg/dl)	10.5 ± 0.05	10.5 ± 0.03
Phosphorus (mg/dl)	3.00 ± 0.60	3.22 ± 0.70
Magnesium (mg/dl)	1.02 ± 0.00	1.05 ± 0.01

Values are mean ± SEM of 3 camels

other animal species, this dose of L-carnitine did not affect lipid profile of dromedary camels. Future planning for further investigations are required to confirm the current results at molecular levels.

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