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Influences of Short -term Aerobic Exercise and Supplementation of Carnitine With or Without Choline on Body Weight, Serum Leptin and Carnitine as Well as Lipid Status In Male Rats

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Abstract

Background: Carnitine is essential for fatty acids translocation, muscles function and exercise performance. Choline is a lipotropic agent that prevents deposition of fat in the liver. The studies concerning the effects of carnitine and choline supplementation with exercise on carnitine status and serum leptin are rare. The aim of the present study was to study the effect of carnitine and its combination with choline, with or without exercise on body and total fat pad (TFP) weights, serum carnitine, leptin, β-hydroxy butyric acid (β-HBA), triacylglycerols (TAG) and Free Fatty acids (FFA). Also, total lipids (TL) and TAG content of TFP and urinary carnitine were investigated.

Material and Methods: 48 male rats were equally divided to the following groups: control (C), carnitine (5 g/Kg diet) supplemented, carnitine plus choline (5 and 11.5 g /Kg diet respectively) supplemented. Half of each group was subjected to short term aerobic exercise on manual treadmill, in which the speed and duration were gradually increased via the course of the experiment, to be 10 m/min for 20 min/day, 5 days/week in the last 2 weeks. Body weights were recorded weekly. After 6 weeks, The 24 hours urine was collected then the fasted rats were sacrificed and blood and the total fat pad (TFP) were collected for analysis.

Results: Carnitine supplementation, tended to decrease body weight, TFP, TAG content and serum FFA, and significantly decreased the TL content, serum leptin, TAG ($P<0.0005$). Carnitine feeding resulted in a significant elevation of serum carnitine, β-HBA and urinary carnitine ($P<0.0005$), compared to sedentary control rats. These values became more pronounced on choline addition to the diet except for serum and urinary carnitine that reversed (i.e. decreased) by choline addition. Exercise intervention resulted in a significant decrease in body weight, TFP, TL content and serum leptin, TAG and FFA. These values were more pronounced in both supplements with exercise, specially serum carnitine. However, exercise caused reduction of urinary carnitine in non-supplemented and carnitine supplemented groups and this was reversed by choline and exercise.

Conclusion: Our study demonstrated that the beneficial effects of carnitine supplements is promoted by choline with or without mild exercise to reduce body weight, body fat, serum leptin and promote fat loss by increasing lipolysis as indicated by increased serum β-HBA. These results may or may not be applicable to humans, so further research is recommended to determine whether similar effects would result in humans or not.

Key words: Carnitine . choline . leptin . β-hydroxy butyric acid . total lipids . exercise

Introduction

L-carnitine, is a naturally occurring substance found in most cells of the body, particularly the brain and neural tissues, muscles, and heart. Carnitine, whose structure is similar to choline, is widely available in animal foods (meat, poultry, fish and dairy products), whereas plants have very small amounts (Iossa *et al*.,2002**)**. A number of studies in a variety of species have shown that, carnitine supplementation influences body composition (Heo *et al*., 2000). It is conceivable

that, carnitine is a co-factor of the enzymatic system involved in long-chain fatty acid transport across the mitoch-ondrial membrane. Also the physiological role of carnitine rose to the hypothesis that, this compound could act as a fat burner by optimizing fat oxidation and a conse-quently reducing its availability for storage (Saldanda *et al.,* 2004). Several studies observed an increase in carnitine utilization during endurance exercise in human (Bross & Hiatt 2004 and Saldanda *et al*., 2004**).**

Choline is a lipotropic agent that prevents deposition of fat in the liver. It is an essential nutrient for humans, providing structure to cell membranes and facilitating transmembrane signaling as well as synthesis and release of acetylcholine. The potential use of choline supplementation for improving physical performance has been reported by many investigators (Decombaz *et al.,* l999 and Leiber *et al*., 2002). On the other hand, choline supplementation to dietary food leads to an increase in fatty acid oxidation and decrease in the total lipids and body weight. These effects will be enhanced by combination of supplementation with mild muscular exercise (Hongu and Sachan 2003). However other study reported that choline-supplemented diet resulted in reduction in the percentage of body fat and increase in the percentage of protein without significantly changing body-weight (Daily *et al.,* l998). Also**,** Dodson and Sachan (2004), reported that, choline supplementation resulted in significant conservation of body carnitine in humans and guinea pigs, this may promote tissue carnitine accretion particularly in skeletal muscle. Combined dietary supplementation of carnitine and choline resulted in an increase in fatty acid oxidation and reduction in the total lipids and body fat. However their combined effect with exercise is controversial (Hongu and Sachan, 2005).

Leptin is a hormone secreted by adipocytes. It may contribute to long-term control of energy balance and body composition by interaction with receptors in the hypothalamus. It circulates in proportion to body fat in both human and rodents (Robert *et al*., 2002 and Flores *et al.,* 2006) .Other sites have been identified as a source of leptin including: skeletal muscle , mammary epithelium, placenta and brain. It plays a central role in the regulation of fatty acid homeostasis, promotion of lipid storage in adipose tissues and fatty acid oxidation in the peripheral tissues. On the other hand, loss of leptin in signaling leads to accumulation of lipid in muscle and loss of insulin sensitivity secondary to obesity (Mcclell *et al.,* 2004**).** It is thought that, the major role of leptin is to relay information to signal transducing receptors in the hypothalamus concerning the status of energy stores and thus aid in reduced feeding its receptors including hypothalamic nuclei (Ahima and Flier, 2000). Leptin receptor mutations cause early obesity in both humans and rats. It is regulated by fat storage with larger adipocytes containing more leptin than smaller ones, and also regulated by insulin level (Saladin *et al*., l999**).** The studies concerning the nutritional factors that influence circulating leptin concen-trations are little specially with exercise. Thus in light of these observations, we proposed that carnitine either alone or with choline supplementation, with or without short term aerobic exercise would alter carnitine status ,serum leptin, body fat and the biochemical marker of fat oxidation, represented by β-HBA, in male rats.

Material and Methods

Chemicals used:

- Carnitine powder was supplemented from Faculty of Science Al-Azhar University as a gift (Sigma, St. Louis, MD).
- Choline powder was purchased from El-Gomhorea-Co for Chemicals.

Experimental design:

Forty-eight adult male albino rats were obtained from Helwan Breeding farm, their initial body weights ranged from 200-215 g. They were fed on ordinary rat show (balanced diet: 22% protein, 5% fat,

69.62% CHO and 3.38% fiber) and water ad-libitum. They were allowed to stay for two weeks in the animal house for accommodation prior to the experiment, and then animals were randomly assigned into three groups (16 each):

- Control group.
- Carnitine supplemented group in a dose of 5 g /kg diet (Hongu and Sachan, 2005).
- Carnitine plus choline supplemented group, 5 g/kg diet and 11.5 g/kg diet respectively (Hongu and Sachan , 2005).
- Half of each group (8 rats) was subjected to short term aerobic exercise program throughout the experiment period (6 weeks).
- Body weights were measured weekly for all groups

Exercise protocol:

Exercise was performed for the exercise groups by a manual treadmill made in the department of physiology in Faculty of Medicine for Girls, Al-Azhar University. The apparatus consists of a metal cage (40 X30X40 cm.) with a movable base. This base made of natural leather rolled on two wheels, one on each side. One of the two wheels is attached to a hand via which the base of the cage is moved manually. The cage is divided longitudinally by a wall, so it can accommodate 2 rats at the same time. The maximum speed reached is 10 meter/min.

All animals in the exercise groups were subjected to run on the treadmill starting with 5min/day during the period of accommodation. At the beginning of the experiment, the running speed and duration were continuously increased during the course of exercise, starting with 5m/min for 5min/day to reach the maximum speed (10m/min for 20min/day) during the last 2 weeks. The exercise sessions were repeated 5 days/week for 6week.

Urine collection:

Twenty four hours urine was collected, by using metabolic cages, one day before sacrificing the animals, for determination of acetyl carnitine content. The urine samples were centrifuged at 1500 x g for 10 min at 4°C,then stored immediately at -70 °C until analysis.

Blood sampling:

At the end of the experiment, all overnight fasting animals were sacrificed by decapitation under light ether anesthesia, and blood samples were collected from carotid artery, centrifuged at 3000 rpm for (10 min). Sera were separated and stored at –70°C until used for analysis.

Total fat pad:

(inguinal, perineal and epididymal) were excised and weighed, rinsed with saline, blotted dry then stored at –70°C until used for determination of triacylglycerols (TAG) and total lipid (TL) contents.

Biochemical analysis:

- Serum leptin concentrations were measured using a commercial radioimmunoassay (RIA)kit (Linco Research, Inc, St. Charles, MO) using a ^{125}I-iodinated human leptin tracer and antihuman leptin rabbit polyclonal antibody (Ma, Zhongmin *et al*., 1996).
- Serum triacylglycerols (TAG), total lipid (TL) and FFA were determined by the methods of Gielgel *et al.* (l975), Ellefson and Caraway (l976) and Novak (l965) respectively.
- Serum β-hydroxybutyric acid (β-HBA) was determined by NAD^+ linked enzymatic reactions ,spectrophotometrically, at 340 nm (Sigma kit no. 310, Sigma, St. Louis Mo.).
- Serum and urine carnitine were measured by the method of Cederblad and Lindstedt (1972) .

Statistical analysis

The data are expressed as means ± SE by using two-way ANOVA to test the effects of supplementation and exercise using SAS (1997***)***. Differences were considered significant at $P < 0.05$.The

superscript letters indicate significant differences among the groups.

Results

Table 1 & Figure 1 demonstrated the changes in body weights gain, total fat pad (TFP) weights and total lipid (TL) and triacylglycerols (TAG) contents of TFP in different groups. Carnitine supplementation resulted in tendency to decrease (P>0.05) in body weight and TFP. These decreases became significant on addition of choline. While both supplements resulted in significant reduction of the TL (P<0.0005) contents and insignificant decrease in TAG contents compared to sedentary control group. Exercise intervention for all groups caused significant decrease in body weights, TFP weights and TL contents and insignificant changes in TAG compared to sedentary rats either supplemented or non-supplemented.

Table 2 & Figures 2 – 5 demonstrated the changes in serum carnitine, leptin, β-HBA, TAG, FFA and urinary carnitine in different groups. Carnitine supplementation resulted in a significant elevation of serum carnitine and β-HBA (P< 0.0005) and a significant reduction in serum leptin and TAG (P<0.0005) with tendency to decrease in FFA. These changes became more pronounced by choline addition and exercise compared to sedentary control rats. On the other hand carnitine feeding increased urinary carnitine (P<0.0005) while choline addition was significantly decreased serum and urinary carnitine. Exercise with carnitine and choline supplementation led to further decrease in serum carnitine but increased urinary carnitine significantly (P< 0.0005) compared to sedentary rats either supplemented or non-supplemented.

Table (1): Mean ± SE of body weight gain, total fat pad weight, and the content of total lipid and triacylglycerols in total fat pad in different groups.

Group	Control		Carnitine supplemented		Carnitine + choline supplemented	
Parameter	**Sedentary**	**Exercised**	**Sedentary**	**Exercised**	**Sedentary**	**Exercised**
Body weight gain (g)	110.6 ± 5.3^{a}	75.3 ± 6.2^{c} P < 0.005	105 ± 6.4^{ab} P > 0.05	70.3 ± 5.8^{c} P < 0.0005	93.5 ± 4.2^{b} P < 0.05	65.8 ± 6.2^{c} P< 0.0005
Total fat pad weight (g)	13.3 ± 0.83^{a}	9.5 ± 0.85^{c} P< 0.0025	11.5 ± 0.81^{ab} P> 0.05	7.4 ± 0.58^{d} P< 0.0005	10.1 ± 0.76^{b} P< 0.005	7.2 ± 0.65^{d} P < 0.0005
Total lipid (g)	9.8 ± 0.82^{a}	6.2 ± 0.28^{b} P< 0.0005	7.1 ± 0.23^{c} P< 0.0005	5.4 ± 0.15^{d} P<0.0005	6.8 ± 0.35^{c} P< 0.0005	5.2 ± 0.31^{d} P< 0.0005
Triacylglycerols (mmol/g. fat)	8.9 ± 0.48^{a}	8.2 ± 0.45^{a} P > 0.05	7.8 ± 0.6^{a} P > 0.05	8.1 ± 0.78^{a} P > 0.05	8.2 ± 0.82^{a} P> 0.05	7.7 ± 0.68^{a} P > 0.05

-Value with different letters in a row differ significantly (P< 0.05).
-The P – value mentioned is compared to sedentary control.

Table (2): Mean ± SE of serum carnitine, leptin, β-HBA, TAG, FFA and urinary carnitine in different groups.

Group	Control		Carnitine supplemented		Carnitine + choline supplemented	
Parameter	**Sedentary**	**Exercised**	**Sedentary**	**Exercised**	**Sedentary**	**Exercised**
Serum carnitine (μ mol/L)	39.2 ± 2.5 [a]	37.3 ± 2.6 [a c] P > 0.05	57.8 ± 2.8 [b] P < 0.0005	62.6 ± 3.1 [b] P < 0.0005	32 ± 2.1 [c] P< 0.0 5	24.9 ± 2.2 [e] P< 0.0005
Serum leptin (μg/ml)	2.31 ± 0.11 [a]	1.9 ± 0.02 [b] P < 0.0005	1.82 ± 0.1 [b] P< 0.0005	1.42 ± 0.05 [c] P< 0.0005	1.61 ± 0.04 [d] P< 0.0005	1.15 ± 0.08 [e] P < 0.0005
β- HBA (μ mol/L)	21.3 ± 2.1 [a]	38.4 ± 3.4 [b] P< 0.0005	53.8 ± 3.2 [c] P< 0.0005	77.8 ± 2.5 [d] P< 0.0005	60 ± 3.6 [e] P< 0.0005	85 ± 2.8 [f] P< 0.0005
Triacylglycerols (mmol/L)	0.79 ± 0.03 [a]	0.72 ± 0.03 [ab] P > 0.05	0.65 ± 0.04 [b] P < 0.0005	0.53 ± 0.03 [c] P< 0.0005	0.46 ± 0.03 [cd] P < 0.0005	0.43 ± 0.02 [d] P< 0.0005
FFA (mmol/L)	275.2 ± 18.3 [a]	285.3 ± 16.2 [a] P> 0.05	245.8 ± 17.3 [a] P> 0.05	169.7 ± 9.5 [b] P<0.0005	179.4 ± 10.3 [b] P<0.0005	142.8 ± 8.5 [c] P< 0.0005
Urinary carnitine (μ mol/L)	145.5 ± 3.8 [a]	65.3 ± 8.6 [b] P< 0.0005	335.8 ± 18.5 [c] P< 0.0005	205.6 ± 11.3 [d] P< 0.0005	55.8 ± 4.2 [b] P< 0.0005	155 ± 3.2 [e] P< 0.0005

-Value with different letters in a row differ significantly (P< 0.05).

-The P – value mentioned is compared to sedentary control.

Fig (1): Mean of body weight gain in different groups.

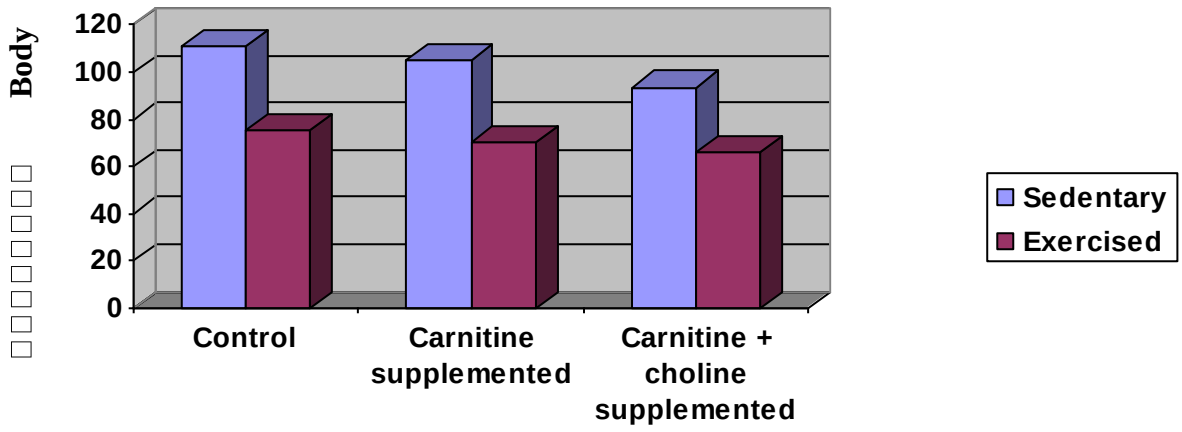


Fig (2): Mean of serum carnitine in different groups.

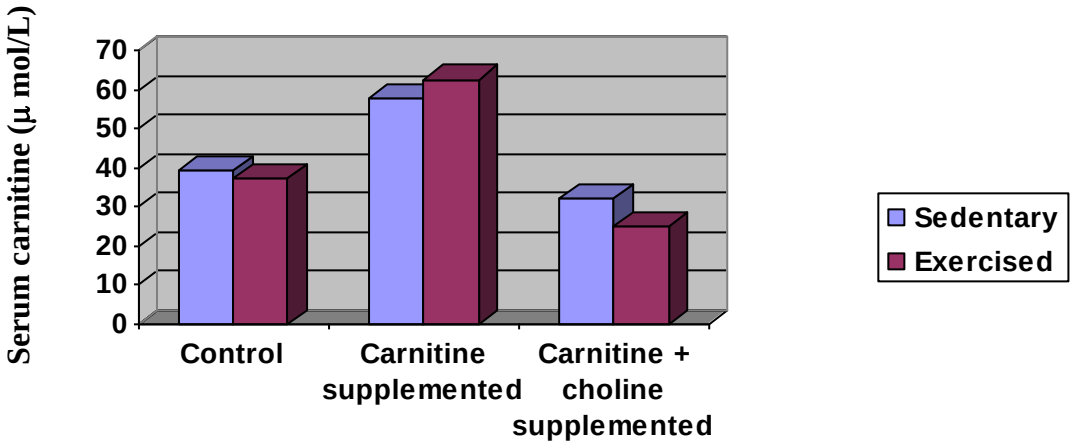


Fig (3): Mean of urinary carnitine in different groups.

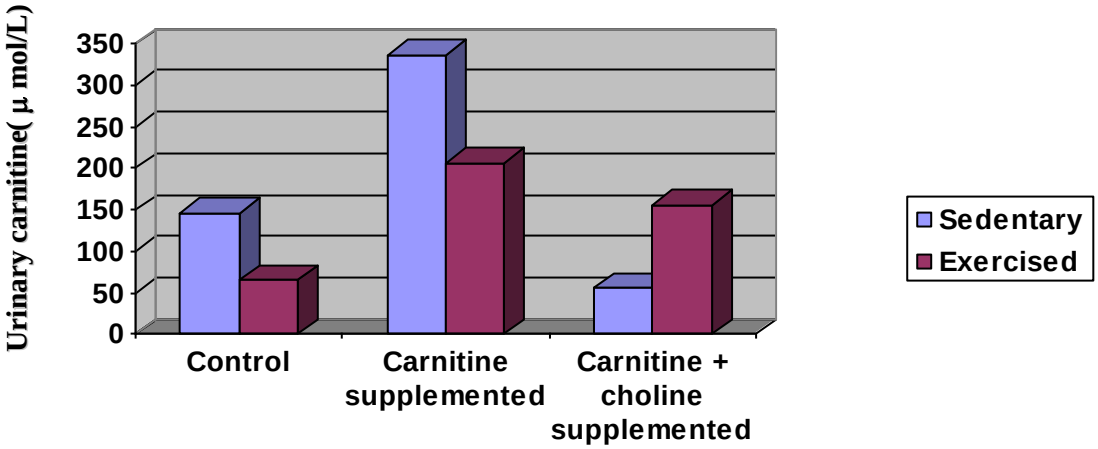


Fig (4): Mean of serum leptin in different groups.

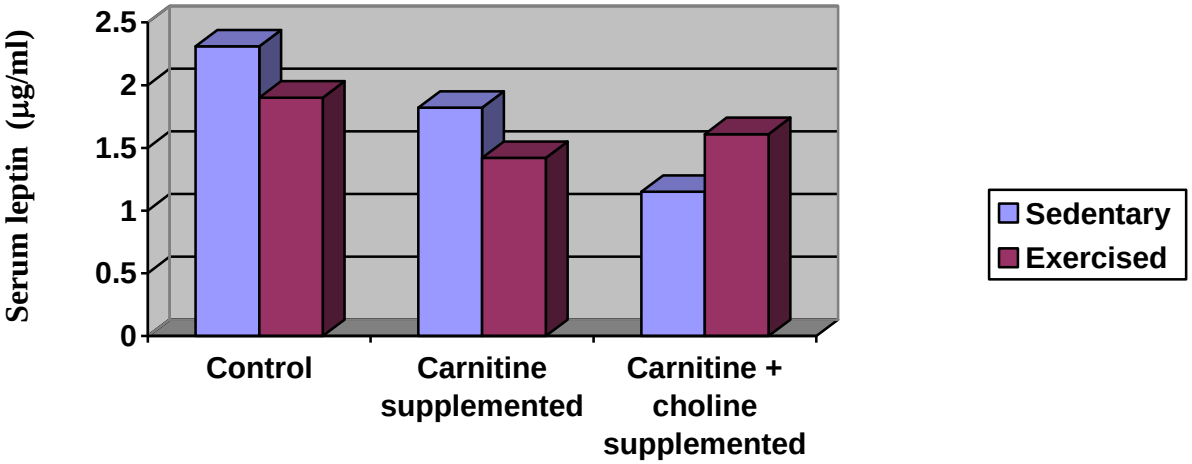
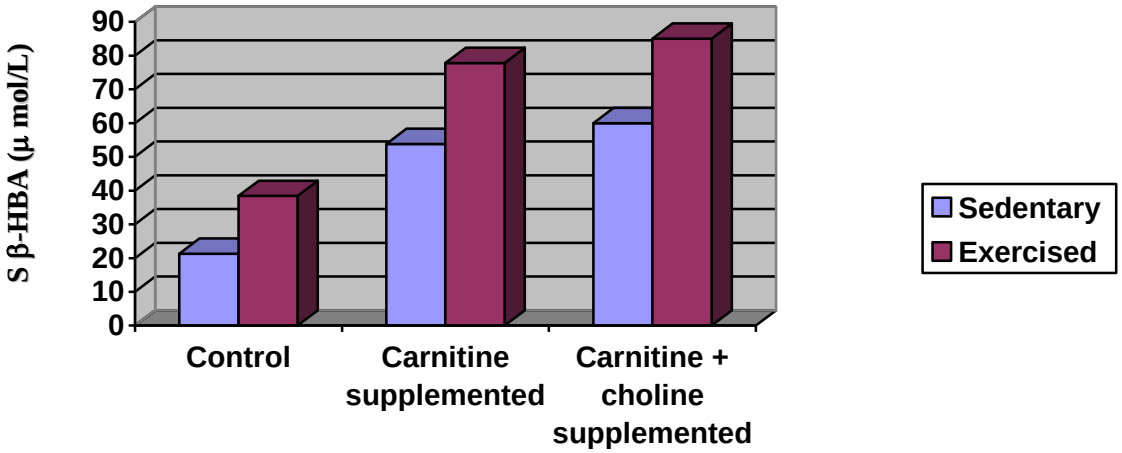


Fig (5): Mean of serum β-HBA in different groups.



Discussion

Exercise training is known to induce physiological adaptations that improve exercise performance and alter patterns of energy substrate utilization to favor fatty acid oxidation. L-carnitine is an essential cofactor for the oxidation of fatty acids under all physiological conditions, includeing exercise (Levin and Meynell, 2006). The current study evaluated the effect of short term exercise and carnitine with or without choline supplementation on body and total fat pad weights, serum carnitine, leptin, β-HBA, TAG and FFA. Also, total lipids and TAG content of TFP and urinary carnitine were investigated.

The present work showed a significant reduction in both body weights and fat pad weights in the rats supplemented with carnitine and those with carnitine combined with choline with or without short term aerobic exercise as compared to the sedentary control. These

findings are in agreement with Hongu and Sachan (2005) who noticed that, physical exercises play a big role in promoting fat oxidation, consequently there is a reduction in the adipose fat mass and weight loss takes place. The combination of choline and carnitine superimposed with mild exercise was hypothesized to enhance oxidation of fat by skeletal muscle and as a consequence reduce the amount of body fat, while diets supplemented with carnitine alone have produced variable responses regarding substrate utilization and muscle fatigue in exercise (Dodson and sachan 2004). On the other hand, the reduction in the body weight, and fat mass may be due to the significant loss of adipose fat mass which enhanced by physical activity (Romijn *et al.,* l999 and Kimura *et al*., 2006). Neumann (2005) found that, diet supplemented with choline and carnitine together induces fatty acid oxidation which was enhanced following physical activity. This may be due to the effect of exercise on the mobilization of FFA by increasesing lipolysis which results from the inhibitory effect of postprandial hyperinsulinemia on lipolysis. Since choline and carnitine have been used as lipotropic agents in a number of studies, it was hypothesized that, their combined effect on fat metabolism may be greater than carnitine alone and may even be enhanced by physical activity where energy demand is increased (Daily and Sachan 2005). Also it was reported that, rats supplemented with choline and carnitine and superimposed by mild exercise for 4 weeks duration, induced a significant reduction of adipose fat when compared with non-exercised animals (Rein *et al*.,2002). In the present work, there was a significant reduction in the total lipid content of fat pads in all exercised rats supplemented with choline and carnitine with or without exercise as compared to the non-exercised- non-supplemented rats. The combination of choline and carnitine with physical activity reduces body fat as indicated by a decrease in fat mass weights and total lipids, this can be attributed to the promotion of energy substrate utilization by supplementation under the condition of exercise. Mild exercise has been shown to promote preferential use of fat for energy needs over and above the basal requirement (Sahtin *et al.,* 2006). Many studies have reported that, physical exercise resulted in reduction in the total and subcutaneous adipose tissues. The reduction in the body fat is supported by decreasing in the endogenous marker of adiposity, leptin, which was significantly lower in the rats supplemented with carnitine with or without choline as well as the exercised rats in our study. In addition, physical activity produce increasing in the phosphorylation activity of several proteins involved in leptin and insulin signal transduction in the combination of choline and carnitine in the hypothalamus (Robert *et al*. 2002 and Flores *et al*., 2006).

In the current study, it was found that, serum leptin showed a significant reduction in the supplemented groups which became more marked after exercise program, these findings were in agreement with the study of Ahima and Flier (2000) who reported the reduction in leptin concentration following physical activity as a result of changes in energy balance, also it is confirmed by more fat mass reduction during exercise, as the leptin concentration is positively correlated with body fat and body weight, and this is pronounced in our study. Most reports of reductions in serum leptin may attribute to circadian rhythms or hemoconcentration. On the other hand, physical activity produces a sufficient energy imbalance. The suppression of leptin level may be counter balanced by feeding, this may explain the reduction in leptin following extreme bouts of exercise such as marathons (Janne *et al.,*2001 and Kraemer *et al.,* 2002). Also leptin concentration can be regulated by insulin level in response to feeding. So during fasting, there is a decline in leptin level following reduction in insulin. Other hormones Such as glucocorticoids, growth hormone and catecholamines, lead to inhibition of leptin production in response to exercise (Commins *et al*., 2001).

In the present study, serum TAG and FFA levels showed a significant decrease in supplemented exercised groups as compared to the resting and exercised rats. On the contrary serum β-HBA showed a significant increase in both supplemented groups and further increased by exercise. These results were in agreement with Lau *et al*. (2001), who reported that both choline and carnitine improve physical performance and muscle function, also altered biochemical markers that were indicative of enhanced fatty acid oxidation to acetate and this is clear in our results, as there were complementary decreases in the serum TAG and FFA in the supplemented groups. Saldanda *et al*. (2004) suggested that, mild exercise enhanced fat utilization as energy substrate. This is reconfirmed in our results in both supplemented groups, but not in control group, as indicated by the increase in the β-HBA levels by exercise beyond that raised by the supplements alone. Couturier et al. (2004) found that, the increase in the β-HBA in the exercised rats may be resulted from the enhanced activity of lipogenic enzymes in adipose tissues, all are decreased following muscular activity. Also, Levin and Meynell (2006) speculated that, muscular activity produced increasing in β-HBA following dietary choline and carnitine supplementation which enhance fatty acid oxidation and mitochondrial events. Borges *et al*. (2005) speculated that, muscular activity resulted in increasing lipolysis, increasing the incorporation of palmitate esterification, decreasing the incorporation of acetate into lipids and diminishes the circulating leptin levels, all these changes can be attributed to the role of pineal gland in the regulation of lipid metabolism which cause physiological balance between lipogenesis and lipolysis during rest and alteration occur with muscular activity.

It is clear from our results that, serum carnitine concentration showed an insignificant increase in the exercised carnitine supplemented group as compared to the sedentary counterpart. On addition of choline to carnitine supplementation, serum carnitine level decreased significantly, and further decreased with exercise as compared to that of the sedentary and exercised controls. Urinary carnitine excretion showed a significant reduction in the exercised control and carnitine supplemented rats as compared to the resting groups. On addition of choline a significant decrease in the urinary carnitine was observed and this was reversed by exercise. This may be due to the increased demand for energy by exercise in choline/carnitine preloaded rats, leads to an increase in the rates of fatty acid oxidation, resulting in sustained loss of acyl groups in urine (Saldanda *et al*., 2004 and Sahtin, 2006). These findings were in line with the study of Neumann (2005) who found that, carnitine contents in the skeletal muscle increased by choline supplementation due to enhancement of carnitine entrance to the skeletal muscles by choline, with subsequent reduction in plasma carnitine levels. Also the decrease in serum carnitine by addition of choline may be due to conservative effect of choline which is in line with the study of Hongu and Sachan (2003), who observed that choline and carintine supplementation over normal dietary intakes altered serum and urinary carnitine profiles in women. Where choline supplementation for 1 wk conserved carnitine, as indicated by decreases in excretion of carnitine in urine similar to that seen in our study. This conservation would be expected to increase the plasma concentrations of carnitine, but it did not in this study. This may be due to a significant shift of carnitine into the tissues of animals supplemented with choline. It is reasonable, therefore, to expect that choline also promotes preferential portioning of carnitine into animal tissues. This explains the relative stability of carnitine concentrations in the plasma compartment of humans and animals in spite of dramatic decreases in urinary carnitine excretion that has been, for most part, accounted for by the tissue pool of carnitine. This is confirmed by Daily *et al*. (1998), who found that choline supplementation increases tissue concentration of carnitine and lowers body fat in Gina pigs.

Conclusion

Our study demonstrated that the beneficial effects of carnitine supplement is promoted by choline with or without mild exercise to reduce body weight, body fat, serum leptin and promote fat loss by increasing lipolysis as indicated by increased serum β-HBA. These results may or may not be applicable to humans, so further research is recommended to determine whether similar effects would result in humans or not.

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تأثير التمرينات الرياضية الهوائية القصيرة مع تعاطي الكارنيتين و الكولين أو بدون الكولين فى الغذاء على وزن الجسم ومستوى اللبتين و الكارنيتين في مصل الدم وحالة الدهون فى ذكور الجرذان

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المقدمة : الكارنيتين مهم لنقل الأحماض الدهنية ولوظيفة العضلات ولأداء الرياضة ، أما الكولين فهو عامل محب للدهون ويمنع ترسيب الدهون فى الكبد. والدراسات التى تخص تأثير الكارنيتين والكولين مع الرياضة على حالة الكارنيتين ومستوى هرمون اللبتين نادرة . ويهدف هذا العمل لدراسة تأثير الكارنيتين أو مع الكولين مع الرياضة أو بدونها على وزن الجسم ووزن الدهون ومحتوى الدهون الكلية وثلاثى أسيل الجليسيرول بها وعلى مستوى الكارنيتين واللبتين وحمض البيتاهيدروكسى بيوتيريك و ثلاثى أسيل الجليسيرول والأحماض الدهنية الحرة بالدم وأيضا على مستوى الكارنيتين فى بول ذكور الفئران.

الطريقة: استخدم فى هذا العمل 48 فأر قسمت إلى المجموعات التالية بالتساوى (16 فأر لكل مجموعة): المجموعة الضابطة، كارنيتين (5 جم/ كجم غذاء)، كارنيتين وكوليين (5 جم و 11,5جم/ كجم غذاء على التوالى) وقد تعرضت نصف كل من هذه المجاميع (8 فئران) لأداء التمرينات الهوائية القصيرة بواسطة جهاز الجرى اليدوى وقد تمت زيادة السرعة ووقت الجرى تدريجيا خلال مدة التجربة وهى ستة اسابيع لتصل إلى 10 متر فى الدقيقة لمدة 20 دقيقة يوميا، 5 مرات فى الأسبوع وهذا فى آخر اسبوعين من التجربة. وقد تم قياس وزن الجسم للفئران إسبوعيا . فى نهاية التجربة تم تجميع بول 24 ساعة لكل فأر ثم جمعت عينات الدم بعد ذبح الفئران لتحليلها. وقد جمعت الدهون التى بالأحشاء ووزنها ثم حفظت لتحليل الدهون بها .

النتائج : تسبب الكارنيتين فى نقص طفيف بوزن الجسم ووزن الدهون ومحتوى ثلاثى أسيل الجليسيرول بها والأحماض الدهنية بمصل الدم. ولكنه أدى إلى نقص ذو دلالة إحصائية عاليه فى محتوى الدهون الكليه ومستوى اللبتين و ثلاثى أسيل الجليسيرول بمصل الدم بينما أدى الكارنيتين إلى زيادة ذات دلالة إحصائية عاليه فى مستوى الكارنيتين وحمض البيتاهيدروكسى بيوتيريك بمصل الدم والكارنيتين فى البول بالمقارنة للمجموعة الضابطة وقد أصبحت هذه القيم أكثر وضوحا بإضافة الكولين للغذاء ماعدا مستوى الكارنيتين بمصل الدم والبول حيث لوحظ نقصهم بإضافة الكولين. وقد تسبب أداء الرياضة فى نقص ذو دلالة إحصائية فى وزن الجسم والدهون ومحتوى الدهون بها ونقص فى مستوى اللبتين و ثلاثى أسيل الجليسيرول والأحماض الدهنية بمصل الدم وهذه القيم كانت أكثر وضوحا فى

مجموعة الكارنيتين مع الكولين والرياضة خاصة مستوى الكارنيتين بمصل الدم. بينما تسببت التمارين الرياضية فى نقص ذو دلالة إحصائية فى مستوى الكارنيتين بالبول فى المجموعة الضابطة والمجموعة المتغذية على الكارنيتين ولكن إضافة الكولين وأداء التمرينات تسبب فى زيادته.

الخلاصة: أوضحت هذه الدراسة أن تأثير الكولين والكارنيتين معا سواء مع ممارسة الرياضة الهوائيه الخفيفة أو عدم ممارستها يؤدى إلى نقص وزن الجسم ووزن الدهون ومستوى اللبتين فى مصل دم الفئران. كما أنه يساعد على التخلص من الدهون بأكسدتها كما إتضح ذلك بزيادة حمض البيتاهيدروكسى بيوتيريك. كما أوضحت الدراسة أن فائدة الكارنيتين تحفزباضافة الكولين و بالرياضة الخفيفة. وتلك الدراسة ربما تنطبق على الإنسان أولا تنطبق ولذلك ننصح بتطبيق تلك الدراسة على الإنسان حتى يتم تحديد ان كانت تسفر عن نتائج مشابهه أم لا.