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Effects of L-carnitine supplementation on physical performance and energy metabolism of endurance-trained athletes: a double-blind crossover field study

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Abstract A double-blind crossover field study was performed to investigate the effects of acute L-carnitine supplementation on metabolism and performance of endurance-trained athletes during and after a marathon run. Seven male subjects were given supplements of 2 g L-carnitine 2 h before the start of a marathon run and again after 20 km of the run. The plasma concentration of metabolites and hormones was analysed 1 h before, immediately after and 1 h after the run, as well as the next morning after the run. In addition, the respiratory exchange ratio (R) was determined before and at the end of the run, and a submaximal performance test was completed on a treadmill the morning after the run. The administration of L-carnitine was associated with a significant increase in the plasma concentration of all analysed carnitine fractions (i.e. free carnitine, short-chain acylcarnitine, long-chain acylcarnitine, total acid soluble carnitine, total carnitine) but caused no significant change in marathon running time, in R, in the plasma concentrations of carbohydrate metabolites (glucose, lactate, pyruvate), of fat metabolites (free fatty acids, glycerol, β-hydroxybutyrate), of hormones (insulin, glucagon, cortisol), and of enzyme activities (creatine kinase, lactate dehydrogenase). Moreover, there was no difference in the result of the submaximal performance test the morning after the run. In conclusion, acute administration of L-carnitine did not affect the metabolism or improve the physical performance of the endurance-trained athletes during the run and did not alter their recovery.

Key words Carnitine · Endurance exercise · Ergogenic · Marathon

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Introduction

The use of carnitine as a potential ergogenic aid is widespread among athletes of any performance category. Several mechanisms have been proposed as to how the administration of carnitine could improve physical performance. It has been suggested that L-carnitine could increase fatty acid oxidation, since it is required for transport of fatty acids across the inner mitochondrial membrane (Fritz 1955). In addition, it has been found that carnitine in skeletal muscle acts as a buffer of free coenzyme A (CoA), which is critical in maintaining normal energy metabolism in skeletal muscle (Friolet et al. 1994). An improvement in physical performance caused by one or both of these mechanisms would require that a sufficient amount of L-carnitine enters skeletal muscle. Hultman et al. (1991) have calculated that a carnitine bolus of 2 g would theoretically increase the muscle carnitine content by only one or two percent. Recently, Brass et al. (1994) have shown that a short-term intravenous administration of carnitine did not change the muscle carnitine content at all. Even after long-term supplementation with $4\ g \cdot d^{-1}$ for 14 days no increase in muscle total carnitine content has been found (Barnett et al. 1994). It has been proposed that carnitine may stabilize the immune system against a downregulation and weakening induced by extreme exercise (Uhlenbruck and van

Mill 1992), for which only an increase in blood carnitine content would be necessary, but also in this case no effects on performance could be expected.

In several studies the effects of an acute or chronic carnitine supplementation on both progressive high-intensity and endurance exercise have been investigated, with some studies showing an increase and others no change in physical performance (for reviews see Ceretelli and Marconi 1990; Wagenmakers 1991). Cooper et al. (1986) have examined the influence of carnitine administration on very long-lasting exercise such as marathon running. However, in that study only few physiological parameters were analysed and the supplementation was provided chronically ($4\ g \cdot d^{-1}$ for 10 days). To the best of our knowledge, the effect of a carnitine bolus on endurance exercise performance has so far not been investigated. Nevertheless, and in spite of some evidence that an acute carnitine administration will not alter muscle performance, this is a common administration pattern among athletes in practice.

The aim of this study was therefore to assess whether a carnitine bolus provided just before and during a marathon run had any influence on running performance and on energy metabolism during and after the run. Moreover, a submaximal performance test in the morning after the run was done to investigate possible effects on the recovery.

Methods

Subjects

Ten endurance-trained male athletes volunteered to take part in this study and gave their written consent after having been informed about the purpose and possible risks of the study. The athletes were chosen on condition that they would be able to perform two marathon runs (maximal running time for one marathon: $3^1/_2$ h) within 4 weeks. As three subjects did not complete both runs, only seven subjects were included in statistical analysis. Some physical characteristics of athletes are shown in Table 1.

Experimental design

The study was designed using the double-blind crossover technique and performed as a field test. The athletes were allocated at random to the sequences of the treatment. A wash-out period of 4 weeks was chosen between the treatments. During the wash-out period the athletes followed their habitual eating and training patterns. The subjects were advised to eat qualitatively the same carbohydrate rich meals for breakfast on the running day and for dinner the day before the run on both occasions. No physical activity was allowed the day prior to the run, and 2 days before the run only slight exercise was permitted.

The running distance was 42.8 km (altitude difference: 800 m). The respiratory exchange ratio (R) was determined on a treadmill before the start and at the end of the run using a half-open system (Oxycon Sigma, Mijnhardt, Bunnik, Netherlands). The athletes were forced to drink 125 ml of sugared tea ($65\ g\ sucrose \cdot l^{-1}$) every 5 km during the run and 500 ml during the recovery period of 1 h. In addition, they were allowed to drink water ad libitum, but food intake was not permitted After both runs, the subjects were advised to eat qualitatively and quantitatively the same food between 1 h after the run and the morning after the run.

The L-carnitine or placebo was administered 2 h before the start (two tablets containing 1-g L-carnitine or placebo), and after 20 km of the run (two tablets containing 1-g L-carnitine or placebo). The L-carnitine and placebo were indistinguishable in shape and taste, and were kindly supplied by Sigma-Tau, Zofingen, Switzerland.

Blood sampling

Blood samples (20 ml) were collected from the antecubital vein into Vacutainer tubes (Becton Dickinson Vacutainer System, Rutherford New Jersey, USA) 1 h before (t_1), immediately after (t_2), 1 h after (t_3), and the morning after the run (t_4). For glucagon analysis 500 KIU Aprotinin (Böhringer Mannheim GmbH, Mannheim, Germany) were added to a glass tube containing 1-ml blood, which was immediately centrifuged for 10 min at 4 °C and 2000 *g*, and the plasma was stored in a glass tube at −20 °C. The remaining 19-ml blood were also centrifuged and the plasma was stored under the same conditions without additive.

Biochemical analysis

The carnitine concentration fractions were analysed using a radioenzymatic assay as originally described by Cederblad and Lindstedt (1972) and modified by Brass and Hoppel (1978). A volume of 1 ml of 6 % (w:w) perchloric acid was added to 1-ml plasma and centrifuged for 2 min at 10,000 *g*. Free carnitine (free Cn) and total acid soluble carnitine (TAS Cn) were determined in the supernatant, and long-chain acylcarnitine (LCA Cn) was analysed in the pellet after alkaline hydrolysis. Total carnitine (total Cn) was calculated as the sum of TAS Cn and LCA Cn, and short-chain acylcarnitine (SCA Cn) as the difference between TAS and free Cn.

The hormone concentrations were analysed using commercially available radio-immunoassay kits (insulin: Kabi Pharmacia

Table 1 Characteristics of the subjects. $\dot{V}O_2$ max Maximal oxygen uptake

Subject no.	Age (year)	Body mass (kg)	Height (cm)	Body Mass Index	$\dot{V}O_2$ max ($ml \cdot min^{-1} \cdot kg^{-1}$)
1	24	69	177	22.0	73
2	39	72	190	19.9	54
3	35	72	182	21.7	60
4	32	72	178	22.7	62
5	48	67	171	22.7	61
6	37	64	173	21.4	68
7	36	67	175	21.9	56
Mean	36	69	178	21.8	62
SEM	3	1	2	0.4	2

Diagnostics, Uppsala, Sweden: glucagon and cortisol: Diagnostic Products Corporation, Los Angeles, USA), and all other metabolites using enzymatic assays at 37 °C on a COBAS-MIRA analyser (Hoffmann-La Roche, Basel, Switzerland).

Submaximal performance test

After the last blood sample the morning after the run, a submaximal exercise test was performed on a treadmill to determine the aerobic-anaerobic threshold. Following a light warming-up the athletes exercised at four intensities on the treadmill at increasing running speeds beginning with 10 $km \cdot h^{-1}$. The increment in speed was 2 $km \cdot h^{-1}$. After having completed 3 min at a given speed, a hyperaemized blood sample (20 µl) was taken from the earlobe and lactate concentration was immediately analysed enzymatically using a LP 400 photometer (Dr. Lange, Zurich, Switzerland). During the test, the heart rate was recorded using a heart rate monitor (Sport Tester PE 4000, Polar Electro, Kempele, Finland). The running speed at the 4-mmol lactate concentration threshold was determined by nonlinear regression.

Statistical analysis

Using the Statgraphics software (STSC Inc., Rockville Maryland, USA) a Friedman two-way analysis by ranks was performed to detect treatment differences between the times t_1 to t_3. In addition, a Wilcoxon paired rank test was performed within the treatment groups with the carnitine fractions. The significance level was set at $P = 0.05$.

Results

Running time and *R*

The running time of the placebo group [198 (SEM 8) min] was not significantly different from that of the carnitine-supplemented group [197 (SEM 9) min]. The mean *R* before the run [placebo 1.00 (SEM 0.01), carnitine 1.00 (SEM 0.00)] and after the run [placebo 0.86 (SEM 0.01), carnitine 0.85 (SEM 0.01)] did not differ significantly between the two groups.

Carnitine fractions

The results of the Cn fractions are listed in Table 2. Carnitine administration was associated with a significant increase in the plasma concentration of all carnitine fractions compared to the placebo group. The within-group analysis in the carnitine-treated group revealed a significant elevation in the plasma concentration of all carnitine fractions immediately after and 1 h after the run compared to the respective values at the start. Similarly, the placebo group showed a significant increase in plasma concentrations of TAS Cn, SCA Cn, LCA Cn, and total Cn immediately after and 1 h after the run, but free Cn had decreased significantly at these two times. In the morning after the run, plasma concentrations of free Cn and total Cn were still elevated in both treatment groups and SCA Cn and LCA Cn had returned to baseline concentrations (t_1). The SCA Cn: TAS Cn ratio had increased significantly immediately after and 1 h after the run and had returned to pre-run values in both groups the morning after the run.

Metabolites and hormones

Plasma concentrations of the different metabolites and hormones, and activities of enzymes are listed in Table 3. Statistical analysis revealed no significant differences between the two groups for concentrations of carbohydrate metabolites (glucose, lactate, pyruvate), fat metabolites (free fatty acid (FFA), β-hydroxybutyrate (BHB), glycerol), hormones (insulin, glucagon, cortisol),

Table 2 Characteristics of the plasma carnitine pool at different times. The *asterisks* denote a significant difference ($P < 0.05$) within the individual treatment group compared to t_1 (Wilcoxon paired rank test). The *P* value shows the level of significance of the Friedman two-way analysis by ranks (the time t_4 was not considered). *Free Cn* free carnitine, *TAS Cn* total acid soluble carnitine, *SCA Cn* short-chain acylcarnitine, *LCA Cn* long-chain acylcarnitine, *Total Cn* total carnitine

Parameter	Treatment	Before run (t_1)		After run (t_2)		After run + 1 h (t_3)		+ 1 day (t_4)		*P* value
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	
Free Cn	Placebo	41.5	2.9	*33.0	1.4	*33.8	3.6	*47.9	2.6	0.005
($\mu mol \cdot l^{-1}$)	Carnitine	45.8	2.0	*70.0	4.9	*70.9	7.4	*58.0	4.2	
TAS Cn	Placebo	47.0	2.6	*56.2	2.4	*51.6	3.1	*56.5	3.7	0.000
($\mu mol \cdot l^{-1}$)	Carnitine	52.8	1.7	*109.4	7.3	*110.6	10.2	*66.0	5.1	
SCA Cn	Placebo	5.6	0.7	*23.1	1.6	*17.9	2.4	*8.6	2.2	0.000
($\mu mol \cdot l^{-1}$)	Carnitine	7.0	0.8	*39.3	4.7	*39.8	4.1	8.0	1.6	
LCA Cn	Placebo	4.4	0.2	*8.4	0.9	*6.2	0.6	*4.4	0.3	0.000
($\mu mol \cdot l^{-1}$)	Carnitine	5.1	0.2	*13.0	1.3	*12.6	1.5	*5.1	0.4	
Total Cn	Placebo	51.4	2.8	*64.6	2.9	*57.8	3.6	*60.9	3.8	0.000
($\mu mol \cdot l^{-1}$)	Carnitine	57.9	1.6	*122.4	8.3	*123.2	11.5	*71.2	5.2	
SCA Cn:	Placebo	0.12	0.02	*0.41	0.02	*0.35	0.04	0.15	0.03	0.074
TAS Cn	Carnitine	0.13	0.02	*0.36	0.03	*0.36	0.03	0.12	0.02	

Table 3 Metabolite and hormone concentrations, and enzyme activities at different times. The time t_4 was not considered for the statistical analysis among the treatments. The *P*-value shows the level of significance of the Friedman two-way analysis by ranks. *FFA* Free fatty acid, *BHB* β-hydroxybutyrate, *LDH* Lactate dehydrogenase *CK* creatine kinase

Parameter	Treatment	Before run (t_1)		After run (t_2)		After run + 1 h (t_3)		+ 1 day (t_4)		*P* value
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	
Glucose	Placebo	5.3	0.4	6.3	0.2	7.3	0 4	4.9	0.3	0.83
($mmol \cdot l^{-1}$)	Carnitine	4.4	0.3	6.1	0.5	7.7	0.5	4.7	0.3	
Lactate	Placebo	1.3	0.1	1.3	0.1	1.5	0.2	1.2	0.2	0.83
($mmol \cdot l^{-1}$)	Carnitine	1.4	0.1	1.4	0 1	1.5	0.1	1.3	0.2	
Pyruvate	Placebo	80	10	90	10	90	10	80	20	0.18
($\mu mol\ l^{-1}$)	Carnitine	80	10	90	10	100	10	110	10	
FFA	Placebo	0.16	0.04	1.57	0.10	1.08	0.12	0.15	0.02	0.51
($mmol \cdot l^{-1}$)	Carnitine	0.16	0.03	1.58	0.12	1.06	0.17	0.16	0.03	
Glycerol	Placebo	0.05	0.01	0.15	0 01	0.07	0.01	0.06	0.01	0.83
($mmol \cdot l^{-1}$)	Carnitine	0.05	0.01	0.13	0.01	0.08	0.01	0.07	0.01	
BHB	Placebo	20	10	100	10	140	30	30	0	0.07
($\mu mol \cdot l^{-1}$)	Carnitine	20	10	170	40	260	70	30	10	
Insulin	Placebo	20.0	5.5	3.6	0 7	6.5	1.4	8.6	1.5	0.65
($\mu U \cdot l^{-1}$)	Carnitine	15.7	3.6	3.0	0.5	6.4	1.5	12.0	3.3	
Glucagon	Placebo	220	20	290	20	210	10	–		0.83
($ng \cdot l^{-1}$)	Carnitine	220	20	290	30	210	10	–		
Cortisol	Placebo	610	50	1310	90	1060	80	480	60	0 51
($nmol \cdot l^{-1}$)	Carnitine	620	70	1270	100	1110	100	410	40	
LDH	Placebo	510	110	1040	60	800	70	720	70	0.83
($U \cdot l^{-1}$)	Carnitine	750	100	1000	110	910	90	880	70	
CK	Placebo	160	20	430	60	420	50	2060	940	0.13
($U \cdot l^{-1}$)	Carnitine	240	60	460	50	480	50	1180	390	

and enzyme activities (lactate dehydrogenase, creatine kinase).

Submaximal performance test

The running speed at the 4-mmol lactate threshold was 16.1 (SEM 0.5) $km \cdot h^{-1}$ versus 15.4 (SEM 0.3) $km \cdot h^{-1}$ ($P > 0.05$) for the placebo and the carnitine group, respectively.

Discussion

The main finding of our study was that no significant changes were detected in plasma concentrations of carbohydrate and fat metabolites, hormones, and in *R* between the placebo and carnitine-treated groups. Since treatment with carnitine did not affect *R* at the end of the marathon, we can assume that substrate utilization during the run was not modified by carnitine. This may explain why no difference in performance, i.e. running time, could be detected between the two treatment groups. Our results concerning *R* are comparable with previous findings of Marconi et al. (1985), who also has not found differences in *R* between placebo and carnitine-treated subjects during a similar endurance exercise test (120 min at 65 % maximal oxygen uptake, $\dot{V}O_2$ max), although the carnitine supplementation was chronic. Other studies using different designs have reported either a decrease in *R* after L-carnitine supplementation (Gorostiaga et al. 1989; Maurer 1987; Wyss et al. 1990) or no difference between placebo and carnitine-treated subjects (Brass et al. 1994; Greig et al. 1987; Oyono-Enguelle et al. 1988; Wyss et al. 1990). Although these studies differ in carnitine administration and type and intensity of the exercise performed, the exact reason for the divergent results remains unclear.

Plasma FFA and glycerol (see Table 3) had an almost identical time course in both treatment groups. As the glycerol concentration in plasma can be considered as an indicator of lipolysis, our results indicated that acute administration of carnitine had no significant effect on adipose tissue lipolysis during exercise. In line with our results, no significant changes in either plasma FFA or glycerol at rest or after exercise have been detected in studies of chronic carnitine supplementation (Föhrenbach et al. 1993; Gorostiaga et al. 1989; Maurer 1987; Oyono-Enguelle et al. 1988; Soop et al. 1988; Wyss et al. 1990). In one study, a significant decrease in percentage of body fat from 12.74 % at 12.18 % has been reported in 17 highly trained athletes during a precompetition training phase after long-term administration of L-carnitine ($30\ mg \cdot d^{-1} \cdot kg^{-1}$

body mass for 3 weeks; Föhrenbach et al. 1993). However, because there was no control group in this study, the possibility cannot be excluded that the decrease in body fat was mainly due to the highly intensive training per se and not to the action of L-carnitine.

A high correlation ($r = 0.92$) between total Cn content in liver and hepatic ketogenesis has been described in rats (McGarry et al. 1975). The same correlation probably exists in human liver, although the rate of ketogenesis in rat liver cells has been shown to be higher than in human liver cells (Emmison and Agius 1988). Similar to the carnitine concentration in plasma, the hepatic carnitine concentration may have been increased after administration of carnitine, since it has been well established that plasma and liver carnitine pools interact closely with each other (Bremer 1983; Ruff et al. 1991). Significantly higher blood BHB concentrations at rest and after 60 min of bicycle exercise at 50 % $\dot{V}O_2$ max have been described after a 28 day-long supplementation with $2\,g \cdot d^{-1}$ L-carnitine (Oyono-Enguelle et al. 1988). In our study, the plasma BHB concentrations were not statistically different between the treatment groups. In any case, since during exercise fatty acid oxidation in the liver accounts only for a minor part of whole-body fatty acid oxidation, no change in R and, more important, no effects on performance would be expected after an increase in liver fatty acid oxidation. As described above, there was no detectable treatment difference in our R result or in the study of Oyono-Enguelle et al. (1988).

Similar to fat metabolites, the exercise-induced changes in plasma concentrations of carbohydrate metabolites did not differ between the two groups (see Table 3). Together with the R data, this indicated that glucose utilization during the run was not altered by carnitine. It has been found that by acting as an acetyl-CoA acceptor and thus reducing product inhibition of the pyruvate dehydrogenase complex (Newsholme and Leech 1994), carnitine might diminish lactate production during supramaximal exercise. However, acute intravenous carnitine administration has been shown not to increase muscle carnitine content (Brass et al. 1994), which would be necessary if lactate production were to be affected. Moreover, even after supramaximal exercise following chronic carnitine supplementation no effect on lactate accumulation in muscle (Barnett et al. 1994) or plasma lactate concentration has been observed (Greig et al. 1987; Marconi et al. 1985; Trappe et al. 1994; Wyss et al. 1990).

No treatment effect was registered in the response pattern of concentrations of the hormones insulin, glucagon, and cortisol (see Table 3). To our knowledge, there are no data available about these hormones during exercise after L-carnitine supplementation.

In summary, an acute L-carnitine load of 2 g before and during a long-distance run did not cause any improvement in running performance. Similarly, there were no significant difference in R and plasma concentration of metabolites and hormones between the treatment groups. Moreover, no treatment effect was observed in the performance at the aerobic-anaerobic threshold during a submaximal test the morning after the run. In line with the failure of acute carnitine administration to increase muscle carnitine content, the present results demonstrated that an acute L-carnitine supplementation had no ergogenic effect and did not improve the recovery in endurance-trained athletes performing a long-distance run.

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