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Effect of L-carnitine supplementation on the sGC/cGMP pathway in vascular relaxing responses from exercised rats

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Background

It has been largely documented the beneficial effects of physical training on the endothelium-derived relaxing response by increasing nitric oxide production and/or its bioavailability to the smooth muscle [1]. L-carnitine (L-Car) has been used as important supplement to regulate body composition associated with lipid metabolism. However, no studies exist investigating the effect of L-Car intake associated with dynamic exercise on the NO-independent sGC/cGMP pathway in the vascular responsiveness in rats. Thus, the aim of this work was to investigate the effect of L-Car supplementation on the NO-independent sGC/cGMP pathway in aortic and mesenteric rings in trained rats.

Methods

Male Wistar rats (344 ± 6 g) were divided into three groups: sedentary (SD), sedentary supplemented (SDS) and trained supplemented (TRS). Animals were trained in a treadmill with an intensity of 70–80% of maximal oxygen consumption, in sessions of 60 minutes, 5 days a week. Run training (RT) was performed simultaneously to L-Car intake (0.2 g/kg daily, given in the drinking water) for 4 weeks. Concentration-response curves were obtained for sodium nitroprusside (SNP) in isolated aortic and mesenteric rings. Plasma SOD activity and catalase levels were measured.

Results

Oral supplementation with L-Car associated with run training provoked a significant reduction in body weight gain (368 ± 13 g) as compared to sedentary groups (SD: 432 ± 9 and SDS: 433 ± 15 g). In aortic rings, the potency (pEC_{50}) for SNP was increased in TRS group as compared to sedentary groups. In mesenteric rings, supplementation with L-Car provokes an increase in the endothelium-independent relaxing response at the pEC_{50} level in sedentary and trained animals (SDS and TRS groups). No changes were seen in the maximal responses to SNP in both preparations. Data are summarized in Table 1. Neither the exercise program nor the chronic supplementation with L-Car affected the plasma levels of catalase (44 ± 14 μM, 69 ± 8 μM and 72 ± 25 μM, for SD, SDS and TRS groups, respectively) and SOD activity (9 ± 4 U/ml, 12 ± 3 U/ml and 11 ± 2 U/ml, for SD, SDS and TRS groups, respectively).

Conclusion

In conclusion, L-Car supplementation associated with physical exercise training was effective to reduce body weight gain in rats. Furthermore, an increase in the relaxing response for SNP in aortic and mesenteric rings from exercise rats treated with L-Car supplementation. These findings suggest that physical exercise-induced shear stress could affect directly the sGC/cGMP signaling pathway in vascular tissues.

Table 1: Potency (pEC_{50}) and maximal responses (E_{MAX}) for sodium nitroprusside (SNP) in mesenteric and aortic rings from sedentary and exercised rats treated with L-Car supplementation for 4 weeks.

Groups	Sodium Nitroprusside			
	Mesenteric		Aorta	
	pEC_{50}	E_{MAX}	pEC_{50}	E_{MAX}
SD	8.31 ± 0.06	103 ± 2	7.90 ± 0.13	109 ± 2
SDS	8.81 ± 0.04*	103 ± 3	7.99 ± 0.13	95 ± 3
TRS	8.58 ± 0.03*	109 ± 4	8.61 ± 0.07*	114 ± 5

Data are means SEM for 4–6 experiments. *, different from sedentary group.

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