CELL BIOCHEMISTRY AND FUNCTION
Cell Biochem Funct 2005; **23**: 369.
Published online in Wiley InterScience (www.interscience.wiley.com). **DOI**: 10.1027/cbf.1247

LETTER TO THE EDITOR

Protective effect of L-carnitine on renal ischaemia–reperfusion injury in the rat

Dear Editor

We read with great interest the article *Protective effect of L-carnitine* on renal *ischemia–reperfusion injury in the rat* in which Görür *et al.* have documented the results of their experimental study regarding the effect of L-carnitine preconditioning on renal ischaemia–reperfusion injury in rats. They have shown that the tissue malondialdehyde (MDA), myeloperoxidase (MPO) and nitrite/nitrate levels were significantly lower in those animals pretreated with L-carnitine prior to induction of renal ischaemia–reperfusion injury than in controls.[1]

We have also previously examined the effects of L-carnitine pretreatment in a rabbit model.[2] In fact, our study was the first in the English literature to submit the novel idea of using L-carnitine in the pretreatment of the ischaemic conditions of the kidney such as transplant surgery, shock states etc.; however, the authors seem to have failed to refer to this study in their article. Our experimental design was similar to that of Görür *et al.*, and we utilized a method of warm renal ischaemia–reperfusion injury, and nephrectomized the contralateral kidneys both to use as controls and to prevent the clearance of lipid peroxidation end-products by the non-ischaemic kidney. Our findings revealed that serum MDA levels were elevated from 1.87 ± 0.72 micromol (pre-ischaemic) to 3.89 ± 1.97 micromol (after reperfusion) in the placebo (saline) group ($p < 0.01$) whereas the pre-ischaemic and post-reperfusion MDA values remained at baseline levels in animals that were treated with L-carnitine (1.63 ± 0.48 vs. 1.68 ± 0.52 micromol respectively; $p < 0.01$). Likewise, tissue MDA levels were significantly elevated in the placebo group after 60 min of ischaemic insult followed by 15 min of reperfusion (468.89 ± 266.06 vs. 640.44 ± 355.08 nmol/grww respectively) while L-carnitine pretreatment significantly reduced post-reperfusion lipid peroxidation (pre-ischaemic and post-reperfusion values 528.89 ± 255.85 vs. 609.56 ± 177.11 nmol/grww respectively; $p > 0.05$). Histopathological examination of the specimen also revealed that glomerular and tubular cellular integrity was well preserved after L-carnitine treatment.

We think that L-carnitine pretreatment is beneficial in terms of reducing the effects of ischaemia–reperfusion injury on kidneys. However, L-carnitine is neither a scavenger nor an antioxidant. The effect of L-carnitine is most possibly due to its ability to transfer fatty acids into the mitochondria where they undergo beta-oxidation and regeneration of coenzyme-A. Therefore, utilizing the alternative energy sources during the ischaemic period helps preserve the membrane integrity and reduce apoptosis.

REFERENCES

1. Görür S, Bağdatoğlu ÖT, Polat G. Protective effect of L-carnitine on renal ischemia–reperfusion injury in the rat. *Cell Biochem Funct* (Online Publication, In Press) 2004; DOI: 10.1027/cbf.1159.
2. Ergun O, Ulman C, Kilicalp AS, Ulman I. Carnitine as a preventive agent in experimental renal ischemia–reperfusion injury. *Urol Res* 2001; **29**: 186–189.

ORKAN ERGÜN AND İBRAHIM ULMAN
Ege University Faculty of Medicine
Department of Paediatric Surgery
Izmir, Turkey