Homocysteine Reduces Energy Metabolism In Amygdala Of Rats: 
Neuroprotective Role Of Creatine

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INTRODUCTION: Homocystinuria is an inborn error of metabolism caused by severe deficiency of cystathionine β-synthase activity, resulting in tissue homocysteine accumulation. Creatine plays a very important role in the energy metabolism. In the present study we investigated some parameters of energy metabolism in amigdala of rats subjected to chronic hyperhomocysteinemia. The effect of creatine was also evaluated. MATERIALS AND METHODS: Wistar rats received daily subcutaneous injection of homocysteine (0.3-0.6 µmol/g body weight), and/or creatine (50 mg/Kg body weight) from their 6th to the 28th day of age. Rats were decapitated 12 h after the last injection and the amygdala was dissected.

RESULTS AND DISCUSSION: Homocysteine administration significantly decreased succinate dehydrogenase, complex IV (cytocrome c oxidase) and Na⁺,K⁺-ATPase activities. In contrast, complex II activity was not changed. This amino acid also altered mitochondrial mass, viability and membrane potential. Creatine prevented the alterations provoked Hcy or caused other effects per se. CONCLUSION: These findings provide insights into the mechanisms by which Hcy exerts its effects. Creatine could be used closely as adjuvant therapy for improvement of symptoms related to an energy deficit in homocystinuric patients.

Keywords: hyperhomocysteinemia, energy metabolism, creatine, amygdala.

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