Oxidative Damage Caused By Ethylmalonic Acid Administration In Rat Heart


1Laboratório de Erros Inatos do Metabolismo, Universidade do Extremo Sul Catarinense, Criciúma, Brazil.
2Laboratório de Fisiopatologia Experimental, Universidade do Extremo Sul Catarinense, Criciúma, Brazil.
3Laboratório de Bioenergética, Universidade do Extremo Sul Catarinense, Criciúma, Brazil.

Patients suffering from short-chain acyl-CoA dehydrogenase deficiency (SCADD) and ethylmalonic encephalopathy (EE) present high concentrations of ethylmalonic acid (EMA) in tissues and biological fluids. These diseases are characterized by cardiomyopathy, which pathophysiological mechanisms are still poorly understood. The aim of the present work was to investigate the effects of acute EMA administration on lipoperoxidation, oxidative protein damage and superoxide dismutase (SOD) activity in rat heart. Thirty-day-old male Wistar rats received three subcutaneous injections of EMA (6 µmol/g, 90 minutes of interval between injections) and were killed 1 hour after the last injection. Control animals received saline in the same volume. Heart was isolated and used for oxidative parameters determinations. It was observed that acute EMA administration increased oxidative damage to lipids and protein, while SOD activity was decreased when compared to control group. Taken together, the results presented herein demonstrate that EMA acute administration caused lipid and protein oxidative damage in heart of young rats, which could collaborate to the heart damage found in patients affected by SCADD and EE.

Keywords: SCAD deficiency; ethylmalonic encephalopathy; ethylmalonic acid; oxidative stress; heart
Supported by: NENASC project (PRONEX program CNPq/FAPESC) and UNESC.