Intracerebroventricular Octanoate Administration Induces Oxidative Damage in Rat Brain

Biella, M.S.¹; Mafioleti, R.L.¹; Rodrigues, L.B.¹; Petronilho, F.²; Ferreira, G.C.¹; Streck, E.L.³; Dal Pizzol, F.²; Schuck, P.F.¹

¹Laboratório de Erros Inatos do Metabolismo, UNESC, SC, Brazil; ²Laboratório de Fisiopatologia Experimental, UNESC, SC, Brazil; ³Laboratório de Bioenergética, UNESC, SC, Brazil.

Introduction: Medium-chain acyl-CoA dehydrogenase deficiency (MCADD) is the most frequent fatty acid oxidation disorder. Patients present tissue accumulation of the medium-chain fatty acid octanoic acid (OA). Clinically, progressive encephalopathy, drowsiness and lethargy that may develop into coma and death are found. Aim: In the present work we investigated the effect of intracerebroventricular OA administration on oxidative stress parameters in cerebral cortex, striatum, hippocampus and cerebral spinal fluid (CSF) of rats. Methods: Animals received a single intracerebroventricular OA injection (1.66 µmol). Control animals received artificial cerebral spinal fluid in the same volume. Animals were killed 1 h after OA administration. Thiobarbituric acid-reactive species (TBA-RS) levels, carbonyl content and superoxide dismutase (SOD) and catalase activities were evaluated. Results: It was observed that OA increased TBA-RS levels and carbonyl content in all tissues tested. In addition, catalase activity was increased only in cerebral cortex and hippocampus. On the other hand, OA administration did not alter SOD activity. Conclusion: Taken together, these data suggest that OA induces oxidative damage in brain. Our results may help to explain, at least in part, the characteristic brain dysfunction observed in MCADD patients.

Keywords: MCAD deficiency, octanoic acid, oxidative damage

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