OXIDATIVE STRESS INDUCTION BY *CIS*-4-DECENOIC ACID IN RAT BRAIN

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Medium-chain acyl-CoA dehydrogenase (MCAD) deficiency is the most frequent disorder of fatty acid oxidation. MCAD-deficient patients present tissue accumulation of the medium-chain fatty acids octanoic, decanoic and *cis*-4-decenoic (cDA) acids. Clinical presentation of MCAD deficiency occurs after fasting and other situations with increased metabolic stress, which precipitate acute symptoms such as lethargy that may develop into coma or even death. In the present work, we investigated the effect of cDA on several oxidative stress parameters in cerebral cortex of young rats, namely chemiluminescence, reduced glutatione levels (GSH), thiobarbituric acid-reactive substances (TBA-RS), total radical-trapping antioxidant potential (TRAP), oxidation of dichlorofluorescein (DCF) and total antioxidant reactivity (TAR). It was verified that cDA significantly induced lipid peroxidation, measured by the increased chemiluminescence and TBA-RS levels. Moreover, DCF oxidation was also increased, in contrast to TRAP, TAR and GSH levels which were markedly decreased by the metabolite. These data strongly indicate a stimulation of lipid peroxidation and free radical production and a reduction of the quantity and quality of the brain non-enzymatic antioxidant defenses and suggest that oxidative damage may contribute, at least in part, to the neurological symptoms observed in MCAD deficiency.

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