COMPARATIVE MITOCHONDRIAL PROTEOME: AN ANALYSIS OF TRANSGENIC HIPERTRIGLYCERIDEMIC MOUSE IN CONTRAST WITH NORMAL TRIGLYCERIDEMIC MOUSE

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Some pathologies of genetic origin like the hypertriglyceridemia, which cause the rise of levels of triglycerides and fatty acids, are related to several cardiovascular diseases. Mice genetically modified to super express the human apolipoprotein CIII show a high mitochondrial resting respiration (state IV). Our proposition was to identify a possible differential expression of mitochondrial proteins related to the change in the functionability of these organelles of transgenic mice. Therefore, using bidimensional electrophoresis (2D) and mass spectrometry we managed to identify three proteins super expressed in the hypertriglyceridaemic animals, among them the Carbamoyl Phosphate Synthase (CPS), which is involved with the metabolism of long-chain lipids and with the regulation of urea secretion and amino acid degradation. Our perspectives are of finding a correlation of the other identified enzymes, metalloproteinase and urate oxidase, in the metabolism of fatty acids and their correlation with the rising of the CPS expression in the hypertriglyceridemic mice. The present work may help in the identification of new molecular targets involved in the regulation of mitochondrial respiration in several states associated to hypertriglyceridemia, like obesity, diabetes and atherosclerosis.

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