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Mitochondria of mice and men: Moderate magnetic fields on ob/ ob mice, human stem

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Abstract

Cell Lines: Implications in Obesity and Fatty Liver.

This paper reviews our research concerning insulin resistance associated with fatty liver which accompanies the metabolic syndrome or diabetes from obesity. Until recently, one hypothesis that has received little attention is that mitochondrial defects are the cause of metabolic syndromes or diabetes 2, fatty liver and insulin resistance. Another hypothesis is that moderate magnetic fields change gene expression. Ob /Ob mice when treated with 0.5 Tesla direct current electromagnetic fields were found to increase their activity, lose weight and fat in a 6 day period. Gene array analysis of human embryonic stem cells in another experiment of 0.23-0.28 T static magnetic fields was conducted. Up-regulation of genes for insulin factors genes, peroxisome proliferative activity receptor were increased, and calcium channel gene and other genes for mitochondrial ribosomal protein S, and uncoupling protein 2. Down- regulation of tumor necrosis factor alpha and interleukin 6 were demonstrated for this transformation. Forkhead transcription factors are also up-regulated at 5 days. Accelerated liver detoxification by moderate magnetic therapy of obesogens that disrupt homeostasis of metabolism of lipids ultimately resulting in obesity is another probable mechanism.

Keywords: Mitochondria, fatty liver, NASH, metabolic syndrome, obesity, diabetes 2, leptin, ATP, insulin resistance, DC electromagnets, oxidative phosphorylation, PPAR, FOXO, UCP2, TNF-alpha, IL6, IGFBP3, INSIG2, LEPR, IRS2, adipocytes, electron transport chain, CACNB2, and MRPS12