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Dysregulation of mitochondrial genetic pathways in injured brain

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Statement of the Problem: Mild traumatic brain injury (mTBI) represents a major health problem in civilian populations as well as among the military service members due to lack of effective treatments and our incomplete understanding about the progression of secondary cell injury cascades resulting in neuronal cell death due to deficient cellular energy metabolism and damaged mitochondria.

Objectives: The aim of this study was to identify and delineate the mitochondrial targeted genes responsible for altered brain energy metabolism in the injured brain.

Study Design: Rats were either grouped into naïve controls or received lateral fluid percussion brain injury (2-2.5 atm) and followed up for 7 days. The expression profiles of mitochondrial-targeted genes across the hippocampus from TBI and naïve rats were examined by oligo-DNA microarrays.

Results: Bioinformatics and systems biology analyses showed 31 dysregulated genes, 10 affected canonical molecular pathways including a number of genes involved in mitochondrial enzymes for oxidative phosphorylation, mitogen-activated protein Kinase (MAP), peroxisome proliferator-activated protein (PPAP), apoptosis signaling and genes responsible for long-term potentiation of Alzheimer's and Parkinson's diseases.

Conclusions: TBI results in impaired regulation of mitochondrial targeted genes in hippocampus responsible for reduced ATP, apoptosis, oxidative stress and increased susceptibility to neurodegenerative disorders.