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Brain proteomics analysis of patients with traumatic brain injury reveals biomarkers for post-TBI complications

Jia Mi, Ganna Shevchenko, Sravani Musunuri, Lars Hillered, Niklas Marklund, Sami Abu Hamdeh and **Jonas Bergquist**
Uppsala University, Sweden

Traumatic brain injury (TBI) constitutes a major cause of death and disability in the most active population in industrialized countries. A serial of medical complications after TBI have been reported, including Posttraumatic Hydrocephalus (PTH), hypertension, and endocrine complications. Moreover, TBI is also considered as an important risk factor for Alzheimer's disease (AD) development. However, how TBI exactly triggers the post TBI complications still remains unclear. In order to provide a more complete picture of TBI pathogenesis related to such progress, we have analyzed brain tissue biopsies from TBI patients for alterations in their proteomes.

In the past decade, mass spectrometry (MS)-based proteomics has become a powerful and reliable technique to analyze complex biological samples. The goal of this study was to identify potential protein biomarkers related to TBI. Two proteomic approaches based on label free quantification MS and stable isotope dimethyl labeling MS were used for quantifying proteins in the brain samples from TBI patients.

Brain proteins were extracted from brain tissue biopsies of TBI patients and controls using 1% n-Octyl-β-D-glucoside lysis buffer. Extracts were delipidated and digested on 3kDa spin filters with endopeptidase LysC-trypsin mixture. Dimethyl isotope labels were used to globally label the tryptic peptides for relative quantification. Individually labelled peptides from samples and controls were combined and analyzed by LC-MS/MS on a 7 T hybrid LTQ FT in duplicate for each sample. The proteins were quantified using MSQuant software. Label-free proteomics analysis was performed on the same LC-MS/MS system and quantified by using MaxQuant and Persus. Our preliminary results revealed a number of proteins with altered expression in brain tissue of TBI patients relative to controls. These proteins represent a wide variety of pathways suggested to be involved in the TCA cycle, calcium signaling, oxidative phosphorylation, apoptosis of neurons, and GNRH signaling. Apart from leading to new insights into the mechanisms that TBI triggers AD, the findings also provide us with possible novel candidates for future therapy target for post-TBI complications.

jia.mi@kemi.uu.se