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Proteomic Approaches to Identify the Mechanism of rapid progression of Alzheimer's Disease

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Recent reports demonstrating one third cases of Alzheimer's disease progressing very rapidly, mimicking prion-based Creutzfeldt-Jakob disease (CJD) and are misdiagnosed. Altered cerebrospinal fluid biomarkers and neuropathology features give some indications, however, still there has been no quantitative study depicting risk factor contributing the fast progression and rapid decline of cognition in AD. In combination with affinity enrichment and high-resolution label free Q-TOF LC-MS/MS analysis, we quantitatively analysed globe wide proteome alteration in thirty cortical brain samples with rapid (rpAD) and slow progressive AD (spAD). A conservative approach of selecting only the consensus results of four normalization methods were suggested and used. Furthermore, we verified differentially expressed proteins at transcriptional and translational level. A total of 79 proteins were shown to be significantly differentially abundant (p-values<0.05, corrected for multiplicity of testing) in rpAD and spAD versus control brain samples (Ctrl). Forty eight proteins were specifically showed different levels specifically in rpAD subjects. Interestingly, in our rpAD dataset selectively, we identified an altered expression level of proteins involved in metabolism of glucose leading to disrupted ATP energy production. We substantiated that the aberrant metabolic networks are a specific phenotype of brain with rapid decline and fast progression of AD.

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