Quantitative proteomics suggests metabolic reprogramming during ETHE1 deficiency - DTU Orbit (08/11/2017)

Quantitative proteomics suggests metabolic reprogramming during ETHE1 deficiency

Deficiency of mitochondrial sulfur dioxygenase (ETHE1) causes the severe metabolic disorder ethylmalonic encephalopathy, which is characterized by early-onset encephalopathy and defective cytochrome C oxidase because of hydrogen sulfide accumulation. Although the severe systemic consequences of the disorder are becoming clear, the molecular effects are not well defined. Therefore, for further elucidating the effects of ETHE1-deficiency, we performed a large scale quantitative proteomics study on liver tissue from ETHE1-deficient mice. Our results demonstrated a clear link between ETHE1-deficiency and redox active proteins, as reflected by down-regulation of several proteins related to oxidation-reduction, such as different dehydrogenases and cytochrome P450 (CYP450) members. Furthermore, the protein data indicated impact of the ETHE1-deficiency on metabolic reprogramming through up-regulation of glycolytic enzymes and by altering several heterogeneous ribonucleoproteins (hnRNPs), indicating novel link between ETHE1 and gene expression regulation. We also found increase in total protein acetylation level, pointing out the link between ETHE1 and acetylation, which is likely controlled by both redox state and cellular metabolites. These findings are relevant for understanding the complexity of the disease and may shed light on important functions influenced by ETHE1 deficiency and by the concomitant increase in the gaseous mediator hydrogen sulfide.

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