

Phosphoproteomic analysis of murine synaptosome in sleep deprivation

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Sleep has been ascribed a critical role in cognitive functioning, with implication in synaptic homeostasis and long-term memory. Acute and chronic stress and total sleep deprivation (SD) impairs memory consolidation, attention, working memory and perception. Despite the wide scientific interest on the topic, there is a lack of systematic investigation of sleep-related changes in the synaptic phosphoproteome.

We isolated parietal cortical synaptosomes of rats after 8 h of total SD by gentle handling and 16 h after the end of deprivation to investigate the short- and long-term effects of SD. Along with protein abundance alterations, MS/MS data of phosphorylation changes in synaptosomes is now available using 6 animals/group and all sample preparation done in replicates. Although most proteins' abundances do not change significantly in SD and recovery sleep, we identified several hundred phosphorylation events that vary between groups. In total 1895 phosphorylation sites were identified.

The data was first analyzed by GO enrichment analysis and pathway analysis. Several altered proteins were found to be involved in synaptic strength regulation, neurotransmitter vesicle trafficking and energy metabolism. The identified protein signaling pathways show the sleep-related synaptogenesis and molecular maintenance. We show that in sleep deprivation, phosphorylation based signalling activates the thyroid hormone synthesis and insuline secretion. Our data also indicates extensive regulation of the Dlg protein family and the dopaminergic pathway which contribute to synaptic plasticity and memory consolidation.