

# Using proteomics in neural regeneration

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## ABSTRACT

The majority of axons still cannot regenerate. This suggests we need to find additional molecules, additional mechanisms. Therefore, much effort has been made to seek molecular pathways that orchestrate neuronal survival and axon regeneration remains a major challenge. One possible reason is that axotomy may impinge on gene transcription as well as protein translation and degradation. Thus, analyzing the transcriptome may not reflect the full scope of injury-induced changes in neurons. Proteins were identified from a range of subcellular compartments, such as the cytoplasm (60%), nucleus (18%), and plasma membrane (14%), and functional classes, such as kinases/phosphatases, transcriptional regulators, or ion channels. As protein abundance is the final readout of gene expression, translation, and degradation, analyzing injury-induced changes in protein abundance may provide a direct assessment of neuronal injury responses. The proteomics approach fills the gap very well and researchers have identified previously unrecognized proteins and pathways involved in nerve regeneration. Proteomic analysis identifies changes correlated to morphological abnormalities in metabolic, contractile and cytoskeletal proteins, deregulation of iron homeostasis, change of Ca<sup>2+</sup> balance and stress response proteins, accompanies by a deregulation of myelin membrane adhesion protein and proteins regulating the neuronal caliber. Overall, scientists identified different proteins through their analysis and are finding some regenerative promise. Using proteomics can be useful to look for new pathways to target in other neurologic disorders such as Alzheimer's disease, frontotemporal dementia, spinal muscular atrophy and amyotrophic lateral sclerosis, as well as sensory nerve injury.

**Key words:** Molecular pathways, Neurologic disorders, Proteomics, Regeneration