Neuroproteomics on the Rise (Part I)

The incidence of neurological disorders is rising markedly with aging. Aged brains become highly prone to neurodegeneration owing to the formation of misfolded protein aggregates and lack of clearance mechanism. Earlier it was thought that accumulation and aggregation of amyloid β (Aβ) peptide is the main driver of Alzheimer’s disease (AD) pathogenesis. But the recent failure of Aβ-targeting clinical trials of drugs led to the mistrust that not only Aβ but also additional pathological mechanisms may play imperative roles. Furthermore, the economic burden of AD patients’ care represents a severe challenge in the health care system. Thus a more diaphanous understanding of AD pathogenesis in humans is essential. In the early stage of the disease, AD diagnosis is diligent and offers a challenging scientific frontline. Therefore, analysis of additional biological markers by using neuroproteomics for the brain aging process would be beneficial to detect disease-specific molecular changes. Similarly, Parkinson’s disease, Huntington’s disease, amyotrophic lateral sclerosis, and other neurological disorders have been increasingly shown to be markedly associated with biologically significant proteomics biomarkers. Advanced neuroproteomics research can not only help in the emergence of new neuroproteomics biomarkers but can also address the qualitative and quantitative sketching as well as functional characterization of patients with neurological disorders. Therefore, this special thematic issue discusses and explores the inherent neuroproteomics aspects associated with various neurological disorders by addressing emerging diagnostic biomarkers and drug targets.

Khurana et al. [1] elucidated the importance of neuroproteomics against several neurological diseases such as Alzheimer’s disease, Parkinson’s disease, multiple sclerosis, epilepsy, as well as psychiatric disorders. The authors attempted to present the current successes and failures of the neuroproteomics approach on the results obtained from different clinical studies that targeted biomarkers associated with neurological disorders.

Narayanan et al. [2] explored the role of neuroproteomics in Alzheimer’s disease research. Elevated levels of amyloid β, total tau, and phosphorylated tau in cerebrospinal fluid have become an important biomarker for the identification of this neurodegenerative disease.

Puranik et al. [3] reviewed the profile of biomarkers, and techniques involved in the discovery of novel biomarkers for early diagnosis of neurodegenerative diseases. The authors stated that identifying the changes in novel protein levels and their functions under the disease conditions is necessary for early diagnosis.

Shaikh et al. [4] presented promising ways of developing competent Alzheimer’s disease therapeutic agents from anti-amyloid aggregating gold nanoparticles. The review highlights some interesting landscapes that might effectively fill the gap in Alzheimer’s disease research.

Mamun et al. [5] emphasized the relationship between the ubiquitin-proteasome system and Alzheimer's disease pathology. The authors also represented the recent therapeutic advancements targeting components of the ubiquitin-proteasome system to combat Alzheimer's disease pathogenesis.

Li et al. [6] focused on the application of iTRAQ and TMT labeling techniques for a better understanding of neurodegenerative diseases. The authors stated that proteomics can shed light on the mechanism by which neurodegeneration manifests as well as aid to establish diagnostic standards and discover new drug targets.

Kumar et al. [7] elucidated the current status of proteomic analysis of Huntington’s disease. This appraisal suggested that analysis of mutation in Huntington’s disease gene can be done efficiently by proteomic analysis, thereby enhancing the effectiveness of treatment.

Bergantin [8] discussed the role of Ca²⁺/cAMP signaling in the interplay between depression and Parkinson’s disease, including the implications for the pharmacotherapy involving Ca²⁺ channel blockers. This appraisal also addressed the fact that Ca²⁺ channel blockers have been demonstrating off-label effects, such as alleviating both parkinsonism and depression symptoms.

REFERENCES


