

Dementia: Neuropathology Based on Changes in Tau Protein

Bedadyuti Mohanty

Faculty of Science and Humanities, Department of Biotechnology, SRM University, Tamil Nadu, India

Abstract:

Dementia is a clinical situation that requires novel practical dependence based on the dynamic memory failure and subjective loss, as its Latin source proposes: an exit from past mental functioning. The occurrence of dementia ascends with age, making it an undeniably common subject among the matured populace. The nature of manifestations among individuals with dementia is more dependent and helpless, both socially and regarding physical and psychological wellness, introducing advancing difficulties to society and to our medical services and hospitals. In spite of the apparently straightforward premise, the clinical analysis of dementia can be troublesome with *de novo* functional disability frequently clouded by physical weakness and misery. Clinical and neurotic basis for the fundamental dementia causing illnesses overlaps remarkably. The development of symptoms takes us into the pathophysiological procedure hamper focused on disorder treatment. An incredible number of research activities are in progress to distinguish potential biomarkers of disease prior its occurrence. Pathological changes of Tau in response to dementia Aggregation of intracellular neurofibrillary tangles (NFT), which comprise of anomalous hyperphosphorylated Tau, is specifically connected with the level of dementia in Alzheimer's disease patients. Various proofs show that the prion-like seeding and spreading of Tau pathology might be the reason of Alzheimer's disease. Previously on various occasions, more prominent research on Tau pathway has uncovered novel areas for the improvement of analysis of specific treatment Tau can manage intracellular transport along the axon. Under physiological situations, very few Tau is identified in dendrites, where it is associated with balancing postsynaptic receptor action. Tau is additionally found in the nucleus. It is supposed that the Tau in the

nucleus maintains the integrity of the DNA by providing support under severe conditions or response. Finally, Tau is demonstrated at minimum levels in astrocytes and oligodendrocytes. Overall considering the Tau activity, the neurological Tau pathology at is significant in AD. eventually the neuronal death. Thus, at early stage of Alzheimer's disease, the diagnosis and removal of Tau proteins and even the seedcompetent monomers (prior to the development of PHFs and NFTs) might be of significantly importance in preventing Tau pathology. Recent advancement over its mechanism study has revealed that these Tau proteins are the significant cytotoxic species (rather than fibrillar aggregates) which disrupts the synaptic function. The disruption of synaptic functions leads the propagation of Tau pathology Presently a number of research investigations on different aspects of dementia have been studied many significant advances have been made to understand its pathology. Dementia is a dynamic form of disorder causing laboratory, social, clinical and financial difficulties. the location where the actual pathology of Tau proteins starts gradually and progressing along the Braak stages III and IV also known as the limbic system and finally it reaches the Braak stages V and VI, which is also the isocortex. The topographical extension of Tau pathology is significantly correlated with the cognitive impairment characteristics and dementia in Alzheimer's disease and it has also been used to categorize the disease into six different Braak stages Dementia is a dynamic form of disorder causing laboratory, social, clinical and financial difficulties. Though the diagnosis of this disorder is clinical, but the successful understanding and treatment can be improved by development of biomarkers. The diseases caused by dementia overlap in their phenotypes and pathophysiology. Tau protein was first introduced over

30 years ago, and was found to be abnormally phosphorylated in the year 1986. Investigation and research over this protein has concluded that it undergoes multiple pathological modifications in of the protein has revealed that Tau undergoes many pathological changes in Alzheimer's disease, where truncation, seeding, hyperphosphorylation, and seeding are the common