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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 patho-physiological procedure hamper focused on disorder treatment. An incredible number of research activities are in progress to distinguish potential biomarkers of disease prior its occurrence.

Keywords: Neuropathology; *Tau* protein; Mental; Alzheimer's disease; Dementia

Introduction

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 treatments [1].

Tau protein is one of the significant microtubule-related proteins in the cerebrum. Whereas, the *Tau* (human) is composed of the microtubule-associated protein *Tau* gene (MAPT), situated on chromosome number 17q21.31. MAPT comprises of many multiple exons (16 in particular). The alternative splicing of exons (number 2, 3, and 10) in the cerebrum generates 6 isoforms of *Tau* comprising of 3 (3-R-*Tau*) or 4 (4-R-*Tau*) C-terminal microtubule-binding repeats and 0 (0-N), 1 (1-N) or 2 (2-N) N-terminal inserts [2].

Discussion

In Alzheimer's disease, affected *Tau* would separate from microtubules, abandoning them with sensitivity against more sensitive proteins such as katanin, by causing significant loss of microtubule mass. Also, *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.

Alzheimer's disease can be characterized on the basis of two predominant factors, where the abnormally hyperphosphorylated *Tau* (intracellular neurofibrillary tangle) are considered as the hallmark. Coeruleus or subcoeruleus complex including the Braak stages I and II (transentorhinal area) are 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 (Figure 1) [3,4].

Conclusion

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. 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 patho-physiology. *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 features.

Future Prospects

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

***Corresponding author:** Bedadyuti Mohanty, Faculty of Science and Humanities, Department of Biotechnology, SRM University, Chennai-603203, Tamil Nadu, India, Tel: 9515636767; E-mail: bedadyutimohanty7@gmail.com

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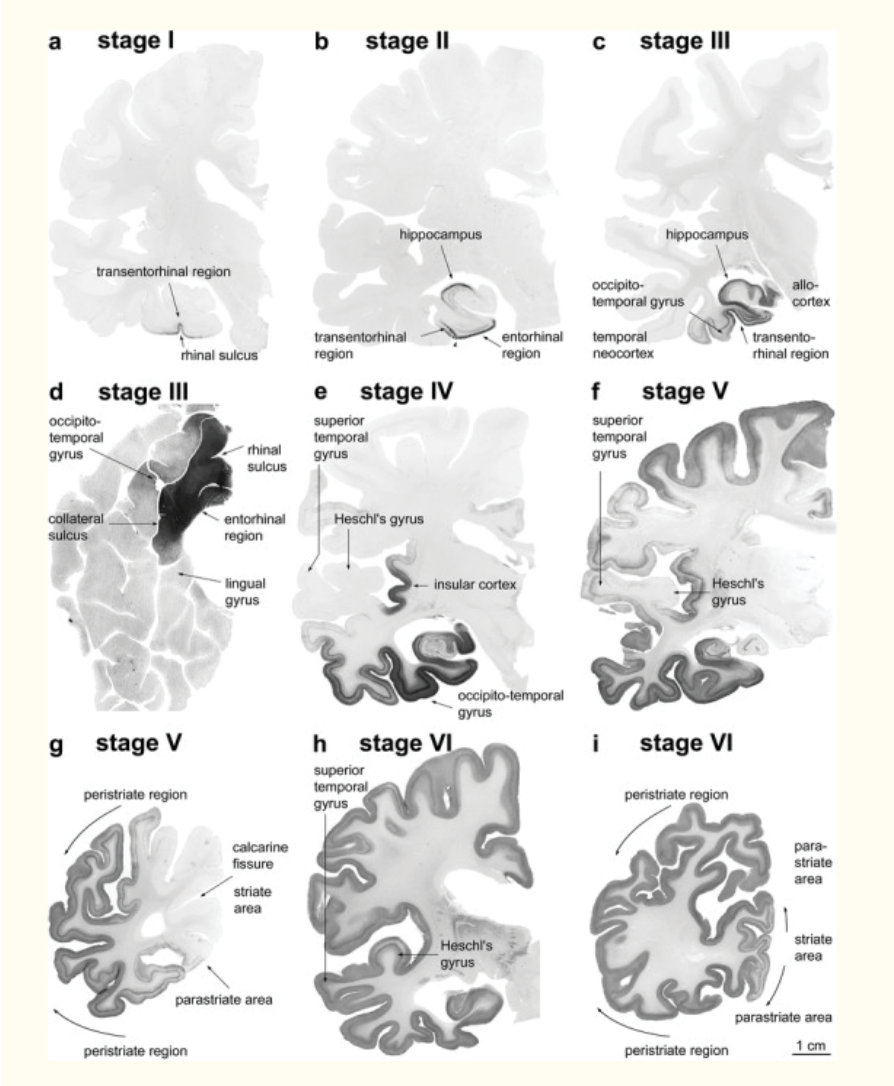

Figure 1: Braak stages from 1-6. Cortical neurofibrillary pathology in hemisphere of the brain (embedded with PEG) and immune-stained for hyperphosphorylated tau [4].

and eventually the neuronal death. Thus, at early stage of Alzheimer's disease, the diagnosis and removal of *Tau* proteins and even the seed-competent monomers (prior to the development of PHFs and NFTs) might be of significantly importance in preventing *Tau* pathology.

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