Neurofibrillary pathology and amyloidogenesis in Alzheimer’s disease: mechanistic insights

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Research Abstract

Alzheimer’s disease (AD) is the most common form of dementia and afflicts 10 percent of population over age 65, 20 percent over age 75 and 50 percent of those age 85 and over. In Canada, more than 300, 000 people currently suffer form AD. This figure is projected to rise to over 500, 000 in 2031. The current yearly economic cost of AD is 10 billion dollars. This tag is estimated to grow to 17 billion dollars in 2031. Currently there are no effective treatments. To develop effective therapies, its cause must be determined. The neuropathological features of AD are progressive neuronal and synapse loss in brain regions associated with cognitive dysfunction and deposition of two defining pathological hallmarks, senile plaques and

neurofibrillary tangles. senile plaques contain beta-amyloid peptide and neurofibrillary tangles are composed mainly of microtubule-associated protein tau. The tau protein becomes abnormally hyperphosphorylated and accumulates leading to the formation of neurofibrillary tangles. Current studies suggest that therapies targeted against both beta-amyloid and tau are essential for treating AD. We have discovered a signaling pathway that controls both beta-amyloid synthesis and tau phosphorylation in the normal brain. This pathway involves a transcription factor called Egr-1. Our studies have shown that the Egr-1 is abnormally increased in AD brain and plays role in the development of pathology. In this proposal, we wish to examine how upregulated Egr-1 in AD brain causes nerve damage and dementia. We will use rat primary neurons and mice expressing human Egr-1 for our studies. Results of our proposed studies will determine step-by step mechanism by which beta-amyloid and tau accumulate leading to the formation of senile plaques and neurofibrillary tangles in AD patients. Determination of such mechanism will open new windows for the development of novel therapies against AD.

Further information available at:

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