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Prion-like amyloid-β oligomers open doors to new molecular mechanisms in Alzheimer's disease

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The aggregation of amyloid-β (Aβ) peptide and its deposition in parts of the brain forms the central process in the etiology of Alzheimer disease (AD). The low-molecular weight soluble oligomers of Aβ (~ 2 - 30mers) have emerged to be the primary neurotoxic agents that have received a substantial attention over the recent years. However, how these toxic agents propagate and proliferate to induce widespread amyloid deposition throughout the brain is far from clear. Pathogenic similarities between Aβ aggregates and those of prion proteins suggest that the proliferation of aggregate lesions may involve a common mechanism based on a 'template-assisted corruptive propagation' process, by which toxic agents amplify themselves by recruiting non-toxic proteins to a template formed by abnormal conformational variants. Recent reports on transgenic mice indicate the infectious nature of both endogenous and synthetic Aβ aggregates and implicate the presence of 'Aβ prions'. Based on several circumstantial evidence, the prion-type mechanism has long been speculated to be conserved among many amyloid-diseases, but remains to be poorly understood. Recently, we generated oligomers of Aβ42 (12-18mers) in vitro, called Large Fatty Acid-derived Oligomers (LFAOs) that show a novel self-propagating property and toxicity towards human neuroblastoma cells. We show that LFAOs are unique Aβ prions that exhibit propagation and neuronal toxicity, and that they can induce 'corruptive propagation' to other homologous amyloidgogenic proteins. Elucidation of prion-like Aβ oligomers presents far-reaching implications not only to AD but also to many neurodegenerative diseases.

Biography

Vijay Rangachari has completed his PhD in Molecular Biophysics at the age of 27 years from All India Institute of Medical Sciences and Postdoctoral studies from Florida State University and Mayo Clinic College of Medicine. He is currently an Assistant Professor of Chemistry and Biochemistry at the University of Southern Mississippi. His research is focused on molecular mechanisms of neurodegenerative diseases, especially Alzheimer's disease and has published more than 25 papers in reputed journals.

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