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Breaking the code of amyloid-β oligomers

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For a century, the cardinal features of Alzheimer's disease (AD), amyloid plaques and neurofibrillary tangles, were thought to underlie this chronic neurological disorder. However, based on the evidence accumulated over the past ten to fifteen years, the toxicity of these lesions has been questioned. Instead the emerging soluble aggregation-intermediate forms of amyloid-beta (Aβ) and tau proteins, which compose plaques and tangles, are now believed to underlie the synaptic and neuronal losses observed in AD. Studies focusing on oligomeric Aβ assemblies have paved the way for other amyloid proteins including tau, alpha-synuclein and the prion protein PrP in the field of neurodegenerative disorders.

This paradigm shift also contributed to complicating even more the putative sequence of biological events responsible for these diseases. The modern view of the amyloid hypothesis suggests the involvement of a multitude of endogenous bioactive Aβ molecules that includes Aβ dimers, trimers, Aβ*56, annular protofibrils and amyloid plaques, as opposed to a single culprit (i.e. plaques). This increased complexity of the problem coupled with a lack of adequate experimental descriptions of the Aβ oligomers used renders interpretation and comparison of the observed phenomena between different research groups arduous and impedes on our progress to better understand the role of Aβ oligomers in AD.

Here, I will try to answer key questions related to Aβ oligomers using our recent data on endogenous Aβ oligomers and argue why we should care about Aβ oligomers and how asking simple questions might generate impactful answers.

Biography

Sylvain Lesne attended college at the Universite de Caen, Basse-Normandie where he graduated with a master's degree in Biochemistry (major in Neuroscience) and a Ph.D. in Molecular and Cellular Biology (major in Neuroscience). He joined Karen H. Ashe's laboratory at the University of Minnesota in November 2002 as a postdoctoral research associate and moved on to becoming a research associate 2 years later. In December 2009, he joined the N. Bud Grossman Center for Memory Research and Care as an Institute for Translational Neuroscience Scholar and tenure-track Assistant Professor (Department of Neuroscience).

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