Title:	Distinct types of amyloid-β oligomers displaying diverse neurotoxicity mechanisms in Alzheimer's disease
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Keywords:	amyloid-β Alzheimer's
Issue Date:	2021
Publisher:	Wiley
Citation:	Journal of Cellular Biochemistry, 122(11), 1594–1608.
Abstract:	Soluble oligomers of amyloid-β (Aβ) are recognized as key pernicious species in Alzheimer's disease (AD) that cause synaptic dysfunction and memory impairments. Numerous studies have identified various types of Aβ oligomers having heterogeneous peptide length, size distribution, structure, appearance, and toxicity. Here, we review the characteristics of soluble Aβ oligomers based on their morphology, size, and structural reactivity toward the conformation-specific antibodies and then describe their formation, localization, and cellular effects in AD brains, in vivo and in vitro. We also summarize the mechanistic pathways by which these soluble Aβ oligomers cause proteasomal impairment, calcium dyshomeostasis, inhibition of long-term potentiation, apoptosis, mitochondrial damage, and cognitive decline. These cellular events include three distinct molecular mechanisms: (i) high-affinity binding with the receptors for Aβ oligomers such as N-methyl- d-aspartate receptors, cellular prion protein, nerve growth factor, insulin receptors, and frizzled receptors; (ii) the interaction of Aβ oligomers with the lipid membranes; (iii) intraneuronal accumulation of Aβ by α7-nicotinic acetylcholine receptors, apolipoprotein E, and receptor for advanced glycation end products. These studies indicate that there is a pressing need to carefully examine the role of size, appearance, and the conformation of oligomers in identifying the specific mechanism of neurotoxicity that may uncover potential targets for designing AD therapeutics.
Description:	Only IISER Mohali authors are available in the record.
URI:	https://onlinelibrary.wiley.com/doi/10.1002/jcb.30141 (https://onlinelibrary.wiley.com/doi/10.1002/jcb.30141) http://hdl.handle.net/123456789/5031 (http://hdl.handle.net/123456789/5031)
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