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The Dynamics of β-Amyloid Aggregation

Stephen C. Meredith

The University of Chicago, Departments of Pathology, Biochemisrtry and Molecular Biology, and Neurology

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 β -amyloid (A β) aggregates are the presumed neurotoxins leading Alzheimer's Disease (AD). Aggregation of $A\beta$ and other amyloidogenic proteins yields polymorphic products, both soluble oligomers and fibrils. This contrasts with normal protein folding, in which a single amino acids sequences leads to a unique protein fold. In this talk, I will discuss the polymorphism and dynamics of βamyloid aggregates. As time permits, I will briefly consider up to four related topics: 1) Structure and polymorphism of fibrils formed by seeding of $A\beta$ solutions by authentic AD brain amyloid. 2) Modelling of these fibrils as aberrant aqueous pores. 3) "Chaotic" aggregation of an internal fragment of $A\beta$, A β 16-34. 4) Formation of A β soluble oligometrs as a second-order phase separation.

