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*Mathematical model for the aggregation of Amyloid-beta.*

Whether Amyloid-Beta ( $A\beta$ ) is the causal factor of Alzheimer's Disease (AD) or a downstream response to some as yet unidentified causative agent, the association of  $A\beta$  aggregation with Alzheimer's Disease means that understanding this process is of considerable importance. Consequently, in this talk, we focus on modeling the process of the aggregation of  $A\beta$ .

In its simplest forms,  $\beta$ -amyloid plaque formation can be described by protein aggregation, involving the misfolding of  $A\beta$  into soluble and insoluble assemblies. Kinetic studies have suggested that the misfolding of monomeric  $A\beta$  has been shown to precede the formation of oligomers, which then serve as seeds for accelerated fibril growth. We will investigate the dynamics of all the stages of the aggregation of Amyloid-Beta, from monomers to dimers, trimers,...oligomers, and fibrils.

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