Aβ(1-40) fibril polymorphism implies diverse interaction patterns in amyloid fibrils

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Abstract	Amyloid fibrils characterize a diverse group of human diseases that includes Alzheimer's disease, Creutzfeldt-Jakob and type II diabetes. Alzheimer's amyloid fibrils consist of amyloid-beta (Abeta) peptide and occur in a range of structurally different fibril morphologies. The structural characteristics of 12 single Abeta(1-40) amyloid fibrils, all formed under the same solution conditions, were determined by electron cryo-microscopy and three-dimensional reconstruction. The majority of analyzed fibrils form a range of morphologies that show almost continuously altering structural properties. The observed fibril polymorphism implies that amyloid formation can lead, for the same polypeptide sequence, to many different patterns of inter- or intra-residue interactions. This property differs significantly from native, monomeric protein folding reactions that produce, for one protein sequence, only one ordered conformation and only one set of inter-residue interactions.
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