Title:	Formation of Heterotypic Amyloids: α-Synuclein in Co-Aggregation
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Abstract:	Protein misfolding resulting in the formation of ordered amyloid aggregates is associated with a number of devastating human diseases. Intrinsically disordered proteins (IDPs) do not autonomously fold up into a unique stable conformation and remain as an ensemble of rapidly fluctuating conformers. Many IDPs are prone to convert into the β-rich amyloid state. One such amyloidogenic IDP is α-synuclein that is involved in Parkinson's disease. Recent studies have indicated that other neuronal proteins, especially IDPs, can co-aggregate with α-synuclein in many pathological ailments. This article describes several such observations highlighting the role of heterotypic protein–protein interactions in the formation of hetero-amyloids. It is believed that the characterizations of molecular cross talks between amyloidogenic proteins as well as the mechanistic studies of heterotypic protein aggregation will allow us to decipher the role of the interacting proteins in amyloid proteomics.
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