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Morphological reorganization of lipid membranes induced by amyloid-beta peptides

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Alzheimer's disease (AD) is one of the most dangerous illnesses leading to the neuronal cell death. It is known that the amyloid-beta (Aβ) peptide plays a key role in this disease, one of the transmembrane fragments of which (Aβ25-35) demonstrates destructive toxic properties. Importantly, the toxic properties of this fragment are manifested upon its interaction with the lipid membrane of cells.
In this work, joint studies of SANS, SAXS, NMR, EM, and MD techniques reveal the membrane breakage when investigating the interactions between Aβ25-35 and model biological membranes mimicking the pre-AD conditions. Namely, it was found that the Aβ25-35 peptide being in the monomeric form incorporated in the lipid bilayer is able to cause a dramatic reorganization between small unilamellar vesicles (SUVs) and bicelle-like structures (BLSs) when transitioning through lipid thermodynamic phases. This effect is explained by the temporal destruction of lipid membranes by Aβ25-35 at the molecular level of a lipid bilayer, whereas it occurs at the superstructure level. In addition, we closely characterize the lipid structure of BLSs and SULVs, as well as localization of peptide molecules.

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