

Abstract Submitted
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Membrane-mediated mechanism of amyloid oligomer toxicity in Alzheimer's Disease. FRANK HEINRICH¹, Carnegie Mellon University, YURI SOKOLOV, JAMES E. HALL, University of California, Irvine, RIMA BUDVYTYTE, GINTARAS VALINCIUS, Institute of Biochemistry, Vilnius, MATHIAS LOESCHE², Carnegie Mellon University — There is strong evidence, that soluble amyloid β ($A\beta$) oligomers, involved in Alzheimer's Disease, are the primary toxic species of $A\beta$, although the mechanism of cell toxicity is very much debated [1]. Neutron reflectivity and electrical impedance spectroscopy assess the structural impact of $A\beta$ (1-42) oligomers and their effect on the electrical properties of a tethered phosphocholine model membrane. Two distinct and reversible peptide – membrane interactions were revealed: At low $A\beta$ concentrations an equal incorporation of $A\beta$ into both lipid leaflets and a compaction of the lipid membrane takes place. $A\beta$ locally lowers the dielectric barrier for ion transport and the activation energy for ion transport through the bilayer remains significantly above that of a water-filled transmembrane pore. At high $A\beta$ concentrations, an additional membrane thinning is observed. [1] D. Eliezer, J. Gen. Physiol. 128:631 (2006).

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