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# Biophysics of neurodegeneration: molecular mechanisms of Alzheimer's disease, towards prevention and cure

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Alzheimer's disease (AD) is a neurodegenerative disease characterized by dementia and memory loss for which no cure or prevention is available. Amyloid toxicity is a result of the non-specific interaction of toxic amyloid oligomers with the plasma membrane. We use biophysical approaches such as atomic force microscopy (AFM), Kelvin probe force
microscopy (KPFM), black lipid membrane (BLM) and surface plasmon resonance (SPR) and other methods to study amyloid aggregation and interaction of amyloid beta (1-42) peptide with lipid membranes.

We tested a set of novel pseudo-peptide inhibitors (potential drug candidates) and showed that they effectively prevent amyloid-amyloid binding on a single molecule level and work well in cellular models to prevent amyloid toxicity. We demonstrated that changes in lipid composition lead to changes in nanoscale membrane structure and alter the membrane-amyloid interactions. We showed that healthy models (HM) are less susceptible to amyloid damage than models mimicking AD-neurons. This possibility of amyloid to recognize the changes in membrane structure and properties opens possibilities to protect the membrane against amyloid toxicity. We studied effects of small molecules that can change the properties of lipid membranes as a way to prevent amyloid induced damage. We found that neuroactive hormone melatonin can efficiently protect the membrane models mimicking early stages of AD but not the late stage of AD. Neuroprotective effects of trehalose and the role of Li salts in neurodegeneration will be also discussed. These findings contribute to a better understanding of the molecular mechanisms of AD and aid to the development of novel strategies for its cure and prevention.

## Keyword-1

## Keyword-2

## Keyword-3

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