MAS17-2017-000022

Abstract for an Invited Paper
for the MAS17 Meeting of
the American Physical Society

Insights into Cross-Linked Amyloid β-Protein Oligomers and Their Role in Alzheimer's Disease[1]

BRIGITA URBANC, Drexel University

Protein misfolding and aberrant protein aggregation are at the core of many age–triggered diseases, such as Alzheimer's, Parkinson's, and Huntington's disease, amyotrophic lateral sclerosis, type II diabetes, systemic amyloidoses, and others. Proteins associated with these diseases do not share any obvious aspects of the primary structure yet they self–assemble into cytotoxic low–molecular weight oligomers and form fibrils with a common cross-β structure. Amyloid β-protein (Aβ) assembly plays a central role in Alzheimer's disease (AD), which is the leading cause of dementia in elderly worldwide. I will describe several computational and experimental approaches that offer insights into formation of Aβ oligomers under oxidative stress conditions occurring in aging brain, which may stabilize Aβ oligomers, inhibit their structural conversion into amyloid fibrils, and prolong their toxic action. Formation and structure of cross-linked Aβ oligomers may hold a key to understanding the basis of Aβ oligomer toxicity and provide clues on how to inhibit their toxic action.

[1]The NSF support through the grant DMR-1429661 is strongly acknowledged.