JOSEPH KLEIN MEMORIAL LECTURE & GRANTING SCHOLARSHIPS OF KLEIN FOUNDATION

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על אלצהיימר, טיעוקים והמ שבריות
(the lecture will take place in Hebrew)

Wednesday, May 17th 2017, at 15:00
Seminar Hall, Los Angeles Building
Gathering & Refreshments at 14:30
ABSTRACT

Our group studies biological self-assembly processes from a physical perspective. Specifically, we study the processes that lead to the formation of a 3D extracellular matrix (ECM) from proteins and polysaccharides in biofilms. The major proteinaceous component of the ECM forms fibrillar appendages that are ‘functional amyloids’. In contrast to amyloid proteins that are related with disease, functional amyloids are not considered harmful but rather, they have a functional role as they provide mechanical stability to biofilms. The formation of amyloid fibers has been extensively studied in the context of neurodegenerative diseases such as Alzheimer’s and Parkinson’s disease. However, very little is known about the mechanisms of functional amyloid formation. Here, we describe the formation of amyloid-like fibers made by two proteins: TasA and TapA. TasA is a structured protein that we purify in the form of stable oligomers. Studying the kinetics of the aggregation process as well as the structure of the aggregates, we try to elucidate the mechanism of the fiber formation. TapA is an auxiliary protein that is necessary for the formation of the fibers. Unlike TasA, there are no reports on the structure of TapA and therefore we firstly study the structure of TapA and only then do we examine its effect on the aggregation of TasA. Last, we study the interaction of the matrix proteins with bacterial membranes. Taking a multidisciplinary approach to study the formation mechanism of the extracellular matrix may lead to the development of new antibiofilm drugs that target the matrix during or after its formation.