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A role of microfluidic flow and gemini surfactants in amyloid aggregation of lysozyme and other proteins

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There is a number of different proteins known, to undergo amyloid aggregation, a specific kind of aggregation being responsible for several severe human diseases. There are neurodegenerative disorders among these diseases, such as Huntington's disease, Parkinson disease, prion diseases, as well as other, like liver cirrhosis or type II diabetes. All amyloid-prone proteins misfold in a similar way, following a common pathway of conformational changes. In quest for an effective drug it is vital to understand the molecular mechanism underlying the process of amyloidogenesis. Parallelly, it is also crucial to screen prospective drugs among various molecules which can inhibit this aggregation.

Microfluidics, an experimental technique dealing with flow of small volumes of samples in channels of small cross sections, has been eagerly used to enhance the scope of traditional research, including amyloid-related studies. Microfluidics offers, among others, low sample consumption, fast analysis, effective mixing of reagents, small droplets formation and their movement control, single nucleation sites tracking, creation of specific flow environment. On the other hand, gemini (dimeric) surfactants, used in our study, are a promising group of compounds in search for substances hampering amyloid aggregation. Their molecules consist of two polar heads linked with a spacer and two hydrophobic tails bound to the heads. Gemini surfactants exhibit good physico-chemical properties, as compared to their monomeric counterparts, such as lower critical micellisation concentration, better solubility, higher surface activity. They are biocompatible and biodegradable, what makes them interesting candidates for testing their inhibitory influence on amyloidogenesis.

In our study we aimed at determining the influence of microfluidic flow, mimicking the flow of proteins in blood vessels, solely on the pace of amyloid aggregation of different proteins, including lysozyme and amyloid beta 1-42 peptide, the latter linked with Alzheimer disorder, in order to coming closer to understanding the molecular mechanism of amyloid aggregation. To reach this aim, we employed a microfluidic system to induce such flow in a glass microchip having one 5-metre long channel and synchrotron radiation small angle X-ray scattering (SR-SAXS), circular dichroism (CD) spectroscopy and transmission electron microscopy (TEM). We also checked how pH and temperature can influence this process. Moreover, we investigated the influence of a number of gemini surfactants on amyloid aggregation of proteins under study. The results of these analysis will be presented on the conference.

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