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Influence of gold nanoparticles on amyloid beta aggregation process

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Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by the gradual deterioration of cognitive abilities, including memory and reasoning, alongside behavioral abnormalities, finally resulting in the patient death [1]. A hallmark of AD pathology is the formation of amyloid plaques, which consist primarily of beta-amyloid (Aβ) protein aggregates in the brain. These toxic Aβ species arise from improper cleavage of amyloid precursor protein (APP), leading to the buildup of harmful peptides that interfere with neuronal communication and function [2]. Beta-amyloid plays a central role in triggering a cascade of neurodegenerative processes, such as dysregulation of calcium signaling, oxidative damage, and inflammatory responses in the brain [3]. Despite ongoing research into the mechanisms of Aβ accumulation, effective therapeutic interventions to halt or reverse AD progression remain elusive. The application of gold nanoparticles (AuNPs) in treating amyloidogenic diseases shows great potential, as the surface chemistry of AuNPs can be precisely tailored to achieve specific binding interactions. This tunability enables AuNPs to serve as effective tools in drug delivery and controlling the aggregation of amyloid proteins, offering a novel approach to managing diseases characterized by amyloid buildup. In this work, three different sized gold nanoparticles were synthesized, and then their effect on the aggregation processes of Aβ 1-42 peptides was studied using P12 (BioSAXS) beamline at PETRA II, DESY (Hamburg, Germany) and fluorescence microplate reader. Gold nanospheres were characterized by UV-Vis spectroscopy, dynamic light scattering and inductively coupled plasma mass spectrometry. Additionally, cytotoxicity tests of fibrils Aβ and Au NPs were performed on the neuroblastoma cell line (SHSY-5Y) from a metastatic bone tumor.

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References:

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