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Role of gold nanorods: Inhibition and dissolution of aβ fibrils induced by near IR laser

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Extracellular plaques of amyloid beta (Aβ) fibrils and neurofibrillary tangles are known to be associated with neurological diseases such as Alzheimer's disease. Studies have shown that spherical nanoparticles inhibit the formation of Aβ fibrils by intercepting the nucleation and growth pathways of fibrillation. In this report, gold nanorods (AuNRs) are used to inhibit the formation of Aβ fibrils and the shape-dependent plasmonic properties of AuNRs are exploited to faciliate faster dissolution of mature Aβ fibrils. Negatively charged, lipid (DMPC) stabilized AuNRs inhibit the formation of fibrils due to selective binding to the positevly charged amyloidogenic sequence of Aβ protein. The kinetics of inhibition is characterized by thioflavin T (ThT) fluorescence, transmission electronic microscopy (TEM), atomic force microscopy (AFM), and attenuated total reflectance Fourier transform infrared spectroscopy (ATR-FTIR). An increase in the aspect ratio of DMPC-AuNR in the range of 2.2-4.2 decreased the fibrils content proportionally. Further, the fibrils content is decreased by increasing the concentration of AuNR for all aspect ratios. As AuNR absorb near-infrared (NIR) light and creates a localized hotspot, NIR laser (800 nm) is applied for 2 min to facilitate the thermal dissolution of mature Aβ fibrils. Majority of Aβ fibrils are disintegrated into smaller fragments after exposure to NIR in the presence of AuNR. Thus, the DMPC-AuNRs exhibit a dual effect: inhibition of fibrillation and NIR laser facilitated dissolution of mature amyloid fibrils. This study essentially provides guidelines to design efficient nanoparticle-based therapeutics for neurodegenerative diseases.

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