Comparative Study of Anti-Amyloidogenic Tannic Acid-Reduced and Tannic Acid-Stabilized Glutathione-Coated Gold Nanoparticles

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Alzheimer's disease, a prevalent neurodegenerative disorder, is characterized by the accumulation of amyloid-beta (Aβ) plaques in the brain, leading to cognitive decline. This study explores a novel therapeutic strategy using tannic acid (TA)-reduced and glutathione (GSH)-coated gold nanoparticles (AuNPs), further stabilized by TA, to inhibit amyloid fibril formation. The study was conducted to determine the synergistic effect when tannic acid stabilized GSH-coated AuNPs, as GSH@AuNP also possesses anti-amyloidogenic properties [1]. Tannic acid was chosen due to its dual role: its bulky molecular structure provides excellent stability to nanoparticles, and its inherent anti-amyloidogenic properties enhance therapeutic potential [2]. By acting as both a reducing and stabilizing agent for GSH-coated AuNPs, TA offers a promising approach to improving nanoparticle stability and efficacy. The synthesized AuNPs were characterized using UV-visible spectroscopy, dynamic light scattering (DLS), field emission scanning electron microscopy (FE-SEM), and X-ray diffraction (XRD) to confirm their stability, size, and crystalline nature. The anti-amyloidogenic potential of these nanoparticles with Hen Egg White Lysozyme (HEWL) was assessed through thioflavin T (ThT) fluorescence assays and confocal microscopy, demonstrating their ability to disrupt amyloid fibril aggregation.
These findings highlight the potential of functionalized AuNPs as promising anti-amyloidogenic candidates. Further investigations could pave the way for their application in drug delivery and neurodegenerative disease treatment.

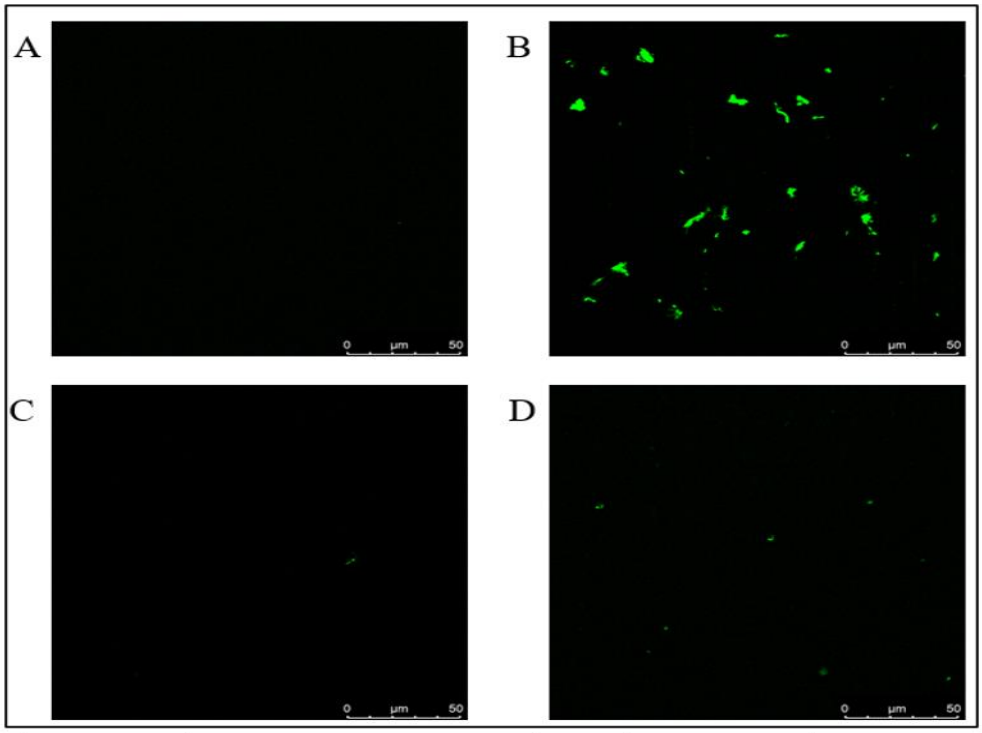

Figure 1 Confocal images of control at '0' hr (A), amyloid formation at 3 hr (B), and images when amyloid formation was inhibited by nanoparticle treatment (C and D)

References

1. Antosova A, Gazova Z, Fedunova D, et al. *Materials Science and Engineering: C*. 2012;32(8):2529-2535.

2. Ono K, Hasegawa K, Naiki H, Yamada M. *Biochimica et Biophysica Acta (BBA) - Molecular Basis of Disease*. 2004;1690(3):193-202.