Photoluminescence Mechanisms of Dual-Emission Fluorescent Silver Nanoclusters Fabricated by Human Hemoglobin Template: From Oxidation- and Aggregation-Induced Emission Enhancement to Targeted Drug Delivery and Cell Imaging

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Abstract

Nicotinamide adenine dinucleotide (NAD) is a critical coenzyme for all living cells. Nicotinamide phosphoribosyltransferase (NAMPT) functions as a key enzyme in the salvage pathway of NAD biosynthesis. Cancer cells have higher rate of NAD consumption and therefore NAMPT is essential for their survival. Thus, we investigated the effect of NAMPT inhibition by miR-206 on breast cancer cell survival. Breast cancer cells were transfected with miR-206 mimic, inhibitor and their negative controls. NAMPT levels were assessed by real-time PCR as well as western blotting. Cell survival assay and quantification of NAD level were performed by using colorimetric methods. Apoptosis assay was performed by labeling cells with Annexin V-FITC and propidium iodide followed by the flow cytometric analysis. Bioinformatics analysis was done to assess whether NAMPT 3′-UTR is a direct target of miR-206 and the results were confirmed by the luciferase reporter assay. NAMPT 3′-UTR was shown to be a direct target of miR-206. miR-206 reduced NAMPT expression at the protein level, leading to a significant decrease in the intracellular NAD level and subsequent decline in cell survival and induction of apoptosis. Targeting of NAMPT-mediated NAD salvage pathway by miR-206 might provide a new insight in the possible molecular mechanism of breast cancer cell growth regulation. This pathway might provide a new approach for breast cancer therapy.