

Determining the potential based electron transfer quenching to develop reagentless nanobiosensor

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Semiconducting nanoparticles have been applied to different fields due to the intense emission, size tunable emission maxima, high quantum yield and photostability of these particles. Protein attached nanoparticles formed through coordination of protein residues to the nanoparticles surface can function as biosensors. Our lab is developing methods using electron transfer to perturb nanoparticles properties for nanobiosensor development. Rat intestinal fatty acid binding protein (IFABP) has been cloned into high expression plasmid *pPROTet.E-133* with and without a carboxy-terminal peptide based on the α -domain of metallothionein (MT) from *Notothenia coriiceps*. We have already demonstrated the His₆-tag mediated nucleation and self-assembly of CdSe nanoparticles in His₆-IFABP which has been characterized by UV-visible spectroscopy and Transmission electron microscopy. A mutation has been made in His₆-IFABP.MT changing the Val to Cys at position 60 and [(5-maleimide-1,10-phenanthroline) (tetraamine)]Ru^{II} complex has been site specifically labeled to cysteine mutant of His₆-IFABP.MT through the maleimide functional group. THDA capped CdSe nanoparticles has been reacted with V60C IFABP.MT to develop metallothionein capped CdSe nanoparticles. Lipid binding has been shown previously to change the hydration of the lipid-binding pocket of IFABP. Using the hydration induced changes in the oxidation potential of [(5-maleimide-1,10-phenanthroline) (tetraamine)]Ru^{II} complex, ΔG for the Ru^{II} induced reduction of CdSe excited state will be varied by lipid binding. Therefore lipid binding will perturb the electron transfer rate and quenching of the CdSe fluorescence. Our research highlights the ΔG dependent electron transfer quenching, examined by the changes in CdSe fluorescence emission intensity, to develop potential based reagentless biosensors.

