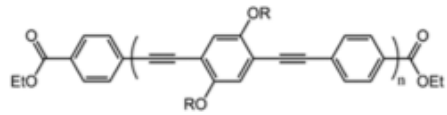


Problem: Protein misfolding and aggregation causes a number of neurodegenerative diseases such as Huntington's, Alzheimer's, and Parkinson's. There is no diagnostic method to detect protein aggregation in vivo. OPEs have been shown to be biocidal due to their ability to generate reactive oxygen species in the presence of visible light, but detection of singlet oxygen generated by OPEs has not been studied.

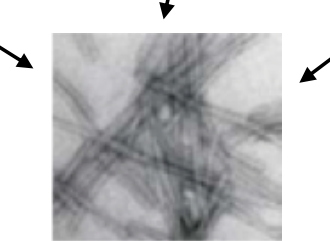
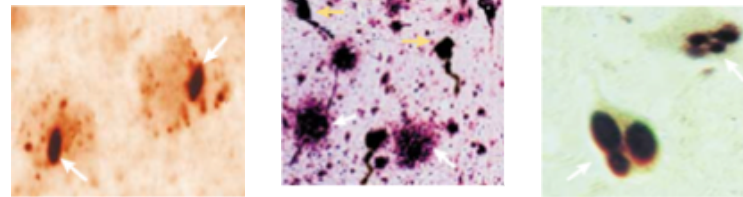
Goal: Test OPE1- and OPE2+ as potential biosensors. Develop a method to monitor the generation of reactive oxygen species.

Method:



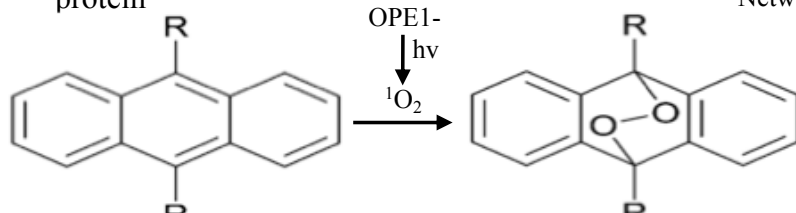
Compound	n	R
OPE1-	1	(CH ₂) ₃ SO ₃ ⁻
OPE2+	2	(CH ₂) ₃ N(CH ₃) ₃ ⁺

Huntington's disease Alzheimer's amyloid plaques and neurofibrillary tangles Parkinson's disease

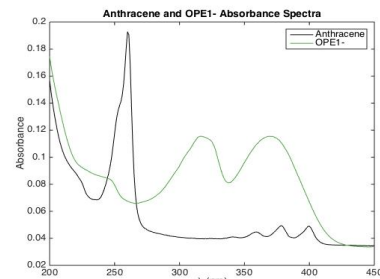


Network of fibrillar polymers

- Huntington's, Alzheimer's, and Parkinson's all share common beta-sheet structure composed of fibrils
- Test OPE1- and OPE2+ as potential biosensors using bovine insulin as model protein

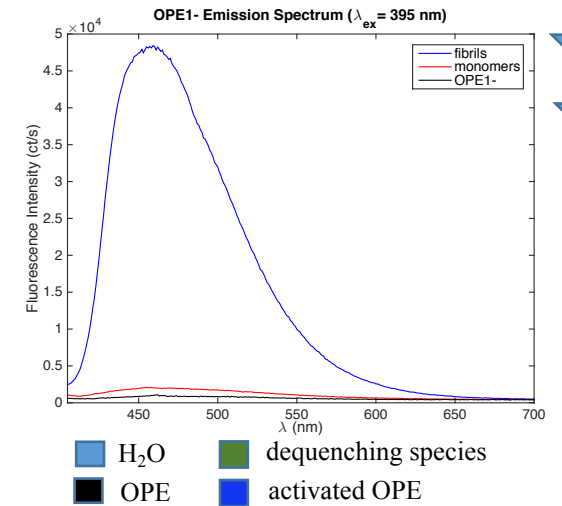
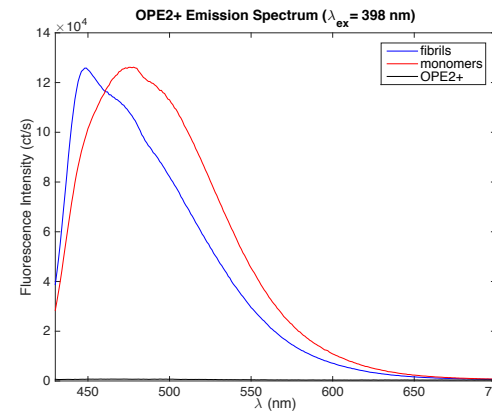


- Use anthracene as a reactive oxygen trap
- Disruption of π -conjugation along backbone is detected by monitoring of characteristic anthracene peak at 261 nm

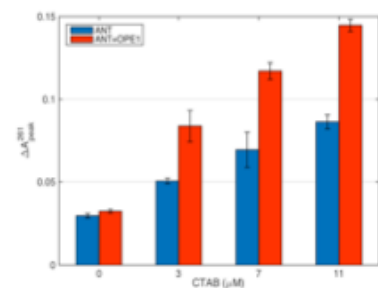
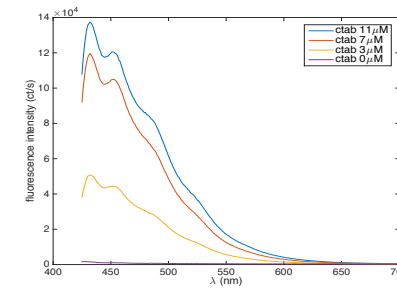
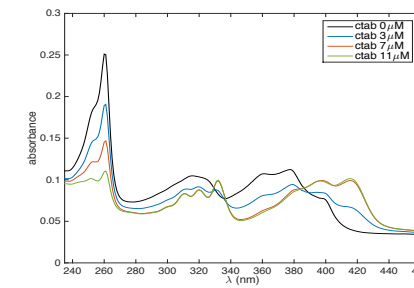


Results:

- Fibrils detected by OPE1- and OPE2+
- Monomers detected by OPE2+ only



- CTAB used a dequenching species
- Increasing CTAB concentration increases anthracene bleaching and fluorescence



Next Steps:

- Investigate the effects of singlet oxygen generation on protein aggregates
- Test OPE1- and OPE2+ against other protein aggregates