

Supporting Information

A Ru³⁺-Functionalized-NMOF Nanozyme as an Inhibitor and Disaggregator for β -Amyloid Aggregates

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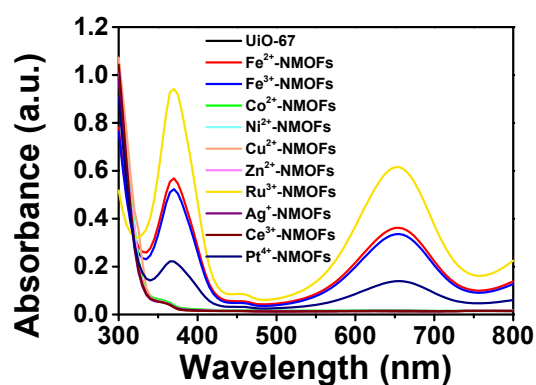


Figure S1. The absorption spectra corresponding to the oxidized TMB generated by a series of Mⁿ⁺-NMOFs with H₂O₂.

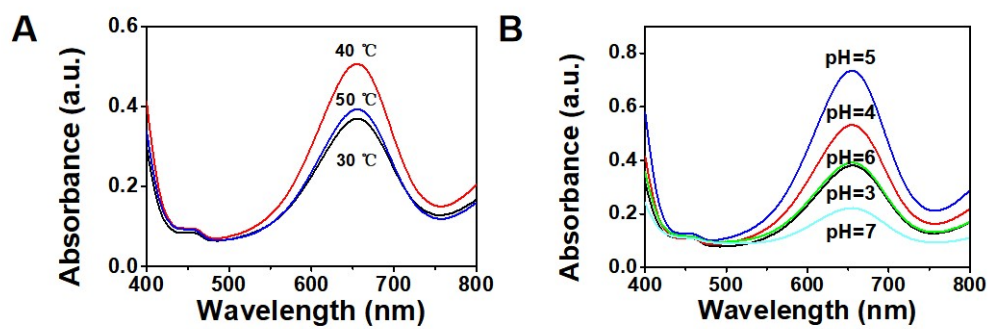


Figure S2. (A) The absorbance of TMB in the presence of Ru³⁺-NMOF + H₂O₂ at 30, 40 and 50 °C. (B) The absorbance of TMB in the presence of Ru³⁺-NMOF + H₂O₂ in HAcO-NaAcO buffer (50 mM, pH=3-7).

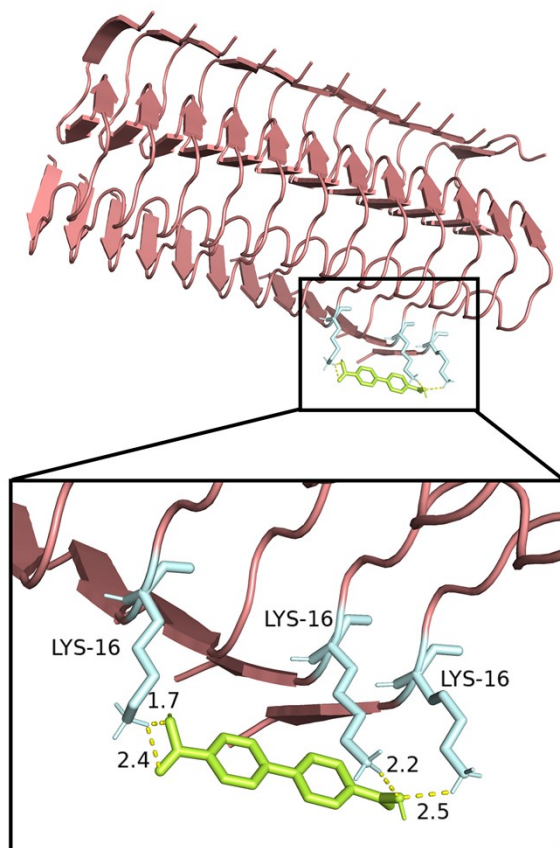


Figure S3. Molecular docking of 2,2'-bipyridyl-4,4'-dicarboxylic acid and A β fibril.

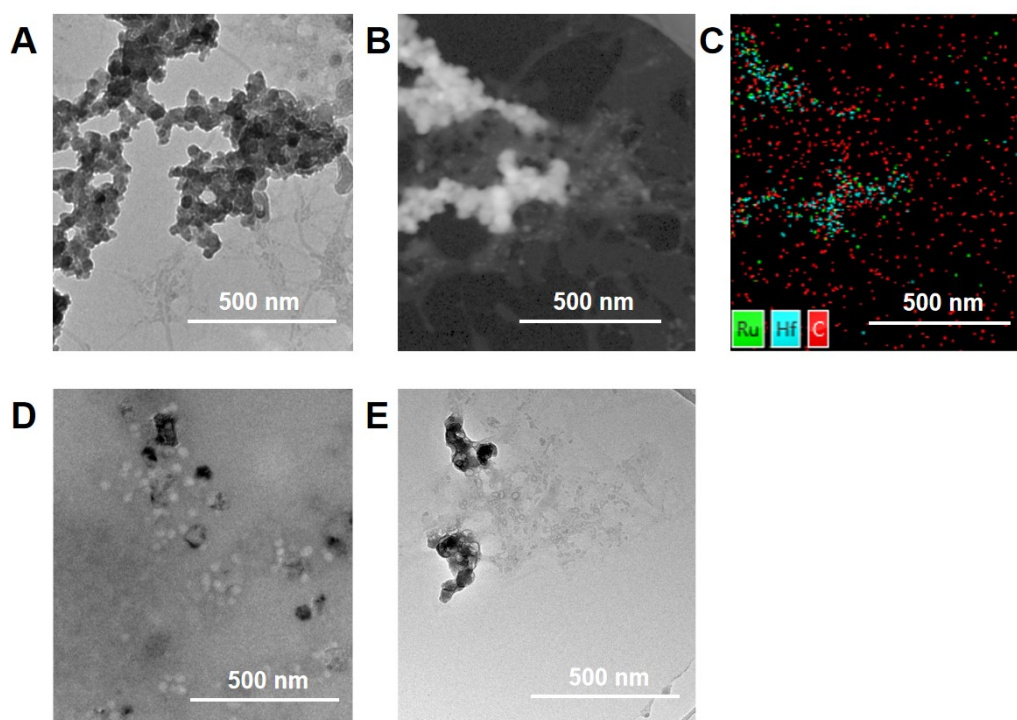


Figure S4. (A) The TEM images of Ru³⁺-NMOF and A β fibrils. (B) TEM dark field of Ru³⁺-NMOF and A β fibrils. (C) The element mapping corresponding to the K-edge signal of Ru³⁺-NMOF and A β fibrils. (D) The TEM images of the product of Ru³⁺-NMOF and A β in the inhibition test. (E) The TEM images of the product of Ru³⁺-NMOF and A β in the disaggregation test.

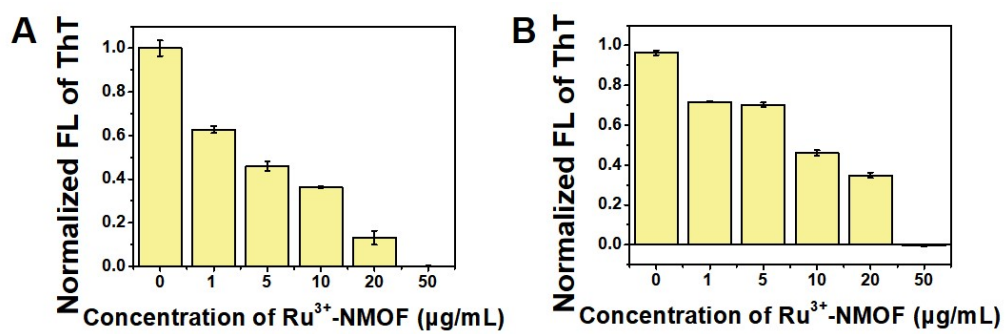


Figure S5. (A) The ThT assay of the final A β products in presence of Ru³⁺-NMOF (1-50 μ g/mL) + H₂O₂ (1 mM) in the inhibition test. (B) The ThT assay of A β fibrils treated by Ru³⁺-NMOF (1-50 μ g/mL) + H₂O₂ (1 mM) in the disaggregation test.

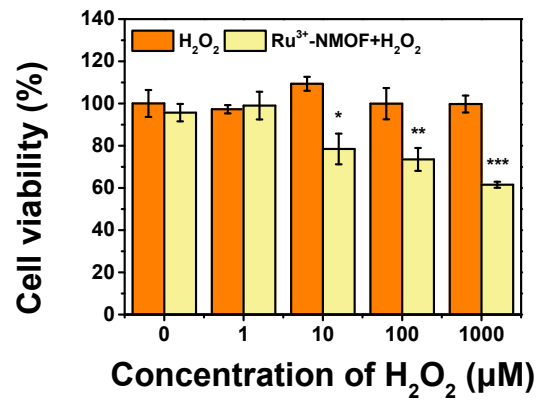


Figure S6. Cell viability in the presence of 1-1000 μM of H_2O_2 with and without 10 $\mu\text{g}/\text{mL}$ of Ru^{3+} -NMOF.

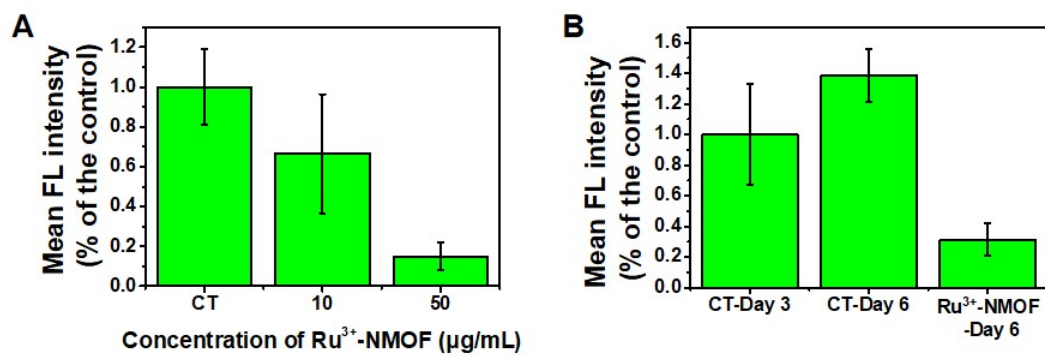


Figure S7. (A) The mean fluorescence intensity of CL2120 treated by Ru³⁺-NMOF (10 or 50 µg/mL) for inhibiting the aggregation of A β . (B) The mean fluorescence intensity of CL2120 treated by Ru³⁺-NMOF (10 µg/mL) for disaggregating A β fibrils.

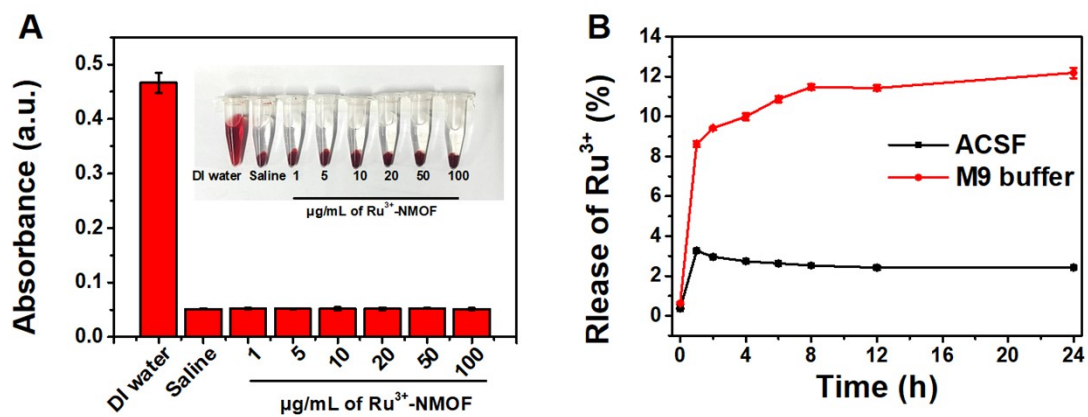


Figure S8. (A) The absorbance ($\lambda=540$ nm) of 5% blood cells with deionized water, saline, and concentrations of Ru^{3+} -NMOF (1, 5, 10, 20, 50, 100 $\mu\text{g/mL}$) in hemolysis test. Insert: the photos corresponding to the groups as noted. (B) The release of Ru^{3+} from Ru^{3+} -NMOF (100 $\mu\text{g/mL}$) at 0, 1, 2, 4, 6, 8, 12, 24 h in ACSF and M9 buffer.