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Supporting information for

Inhibitory effects of NAMI-A-like ruthenium complexes on prion neuropeptide fibril formation

Xuesong Wang, Dengsen Zhu, Cong Zhao, Lei He, Weihong Du*

Department of Chemistry, Renmin University of China, Beijing 100872, People's

Republic of China

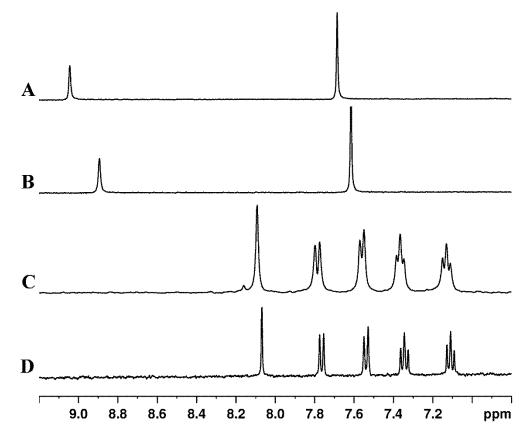


Figure S1. The ¹H-NMR spectra of the complexes KP418 (A), NAMI-A (B), KP1019 (C), and KP1019-2(D).

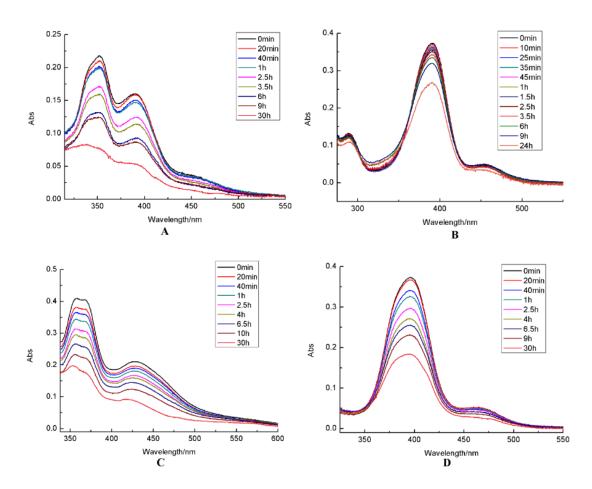


Figure S2. The UV-vis spectra of NAMI-A-like complexes carried out in aqueous solution. A. KP418, B. NAMI-A, C. KP1019, D. KP1019-2.

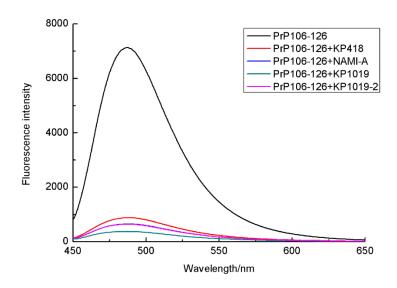


Figure S3. Evaluation of the ability of KP418, NAMI-A, KP1019, and KP1019-2 to inhibit the aggregation of PrP106–126.

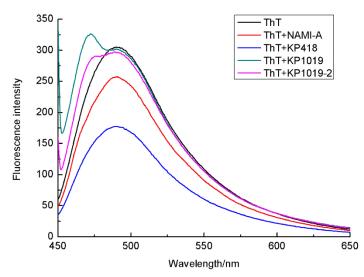


Figure S4. The ThT spectra carried out in the presence and absence of ruthenium complexes. Compared with fluorescence intensity of PrP106-126, the FL intensity of metal complex is small enough to be ignored.

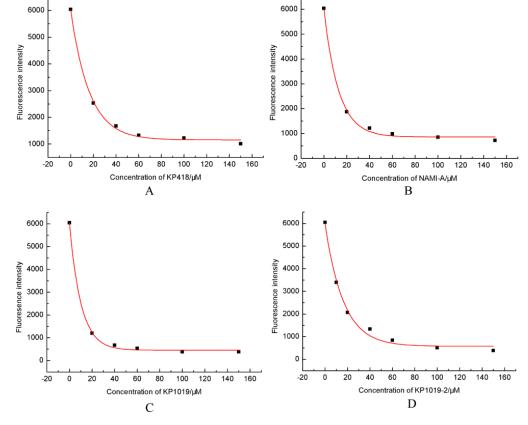


Figure S5. The ability of ruthenium complexes to inhibit the aggregation of PrP106–126, KP418 (A), NAMI-A (B), KP1019 (C), and KP1019-2 (D) measured by ThT fluorescence assay. The concentration of the peptide was 100 μ M and the IC⁵⁰ values were summarized in Table 2.

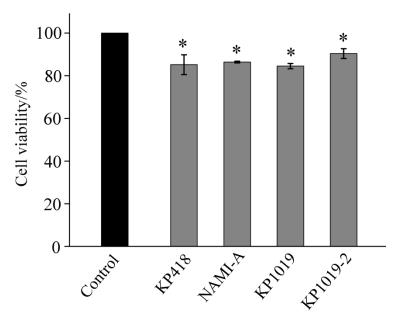


Figure S6. Effects of ruthenium complex on the viability of human SH-SY5Y cells determined with a MTT assay. It means that ruthenium complex have a relative low cytotoxicity. $^*P < 0.01$ versus control group.