Supporting materials for

Influence of gold-bipyridyl derivants on aggregation and disaggregation of the prion neuropeptide PrP106–126

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Figure S1. ¹H NMR spectra of gold complexes in 9:1 H₂O/d₆-DMSO solvent at pH 5.8, 298 K. [Au(Me₂bpy)Cl₂]Cl (A); [Au(t-Bu₂bpy)Cl₂]Cl (B); [Au(Ph₂bpy)Cl₂]Cl (C).

Figure S2. ESI-MS spectra of 50 μ M PrP106–126 in the presence of equivalent amounts of [Au(Ph₂bpy)Cl₂]Cl with pH value at 5.8 (A) and 7.0 (B).

Figure S3 The portion of downfield NMR spectra of $[Au(Me_2bpy)Cl_2]Cl$ in the absence (A) and presence of PrP106-126 (B).

Figure S4 The portion of downfield NMR spectra of $[Au(t-Bu_2bpy)Cl_2]Cl$ in the absence (A) and presence of PrP106-126 (B). The peak at 8.04 ppm is added in (B) due to its overlap with the peaks from peptide.

Figure S5 The portion of downfield NMR spectra of $[Au(Ph_2bpy)Cl_2]Cl$ in the absence (A) and presence of PrP106-126 (B).

Figure S6. The abilities of metal complexes $[Au(bpy)Cl_2]PF_6$ (blue), $[Au(Me_2bpy)-Cl_2]Cl$ (black), $[Au(t-Bu_2bpy)Cl_2]Cl$ (red) and $[Au(Ph_2bpy)Cl_2]Cl$ (green) to inhibit the aggregation of PrP106–126 measured by ThT assay. The concentration of the peptide was 100μ M.

Figure S7. The neurotoxicity of gold complex determined by MTT assay. The data represented the average of four experiments.