

Morphological and *in vitro* evaluation of programmed cell death in MCF-7 cells by new organoruthenium(II) complexes

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Supporting Information

The IR spectra of the [**HL**¹-**HL**⁴] ligands and its complexes provide information about the metal ligand bonding. A strong absorption at 1556-1637 cm⁻¹ in the ligands [**HL**¹-**HL**⁴] is assigned to the presence of $\nu_{(C=N)}$ group. This band undergoes a negative shift of 38-42 cm⁻¹ in all the complexes, which can be attributed to the donation of lone pair of the nitrogen to the metal ion during the coordination.^{1,2} The ligand may exist in thione-thiol tautomerization since it contains a thioamide (-NH-C=S) functional group. The absence of the $\nu_{(S-H)}$ stretching frequency in the region 2500-2600 cm⁻¹ and the presence of $\nu_{(N-H)}$ stretching frequency in the region 3231-3434 cm⁻¹ in the IR spectrum of the ligand indicate thione form in the solid state. This is further inferred from the presence of a strong band in the region 826-863 cm⁻¹ due to the $\nu_{(C=S)}$ stretching frequency which completely disappeared in the complex **4** and a new band appeared at 740 cm⁻¹ corresponding to $\nu_{(C-S)}$ indicating the enolisation of NH-C=S group and subsequent coordination through the sulphur atom.^{3,4} However in complexes **1-3**, the absence of the $\nu_{(S-H)}$ stretching frequency and presence of $\nu_{(N-H)}$ stretching frequency at 3011-3057 cm⁻¹ indicate the involvement of the thione sulphur in the coordination rather than thiolate.^{5,6} The electronic spectra (Fig. S1) of the complexes have been recorded in CH₂Cl₂ and they displayed two to three bands in the region around 242–349 nm. The bands appeared in the region 242–295 nm have been assigned to intra ligand transition,⁷ the bands around 309–349 nm were due to LMCT (s→d).⁸

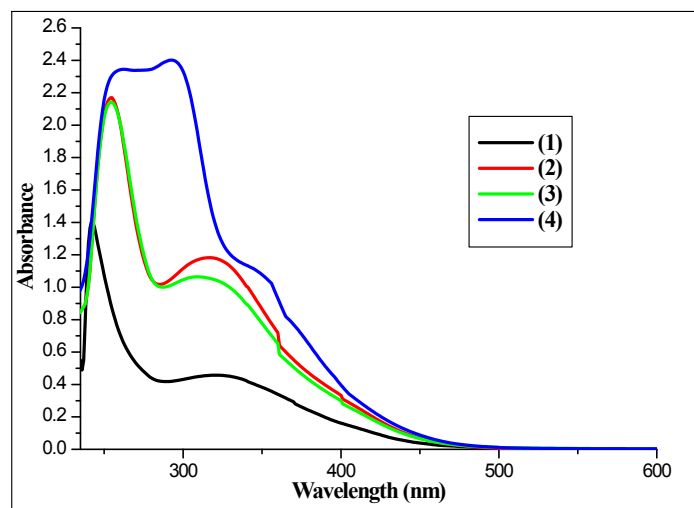


Fig. S1. Electronic absorption spectrum of complexes (1-4)

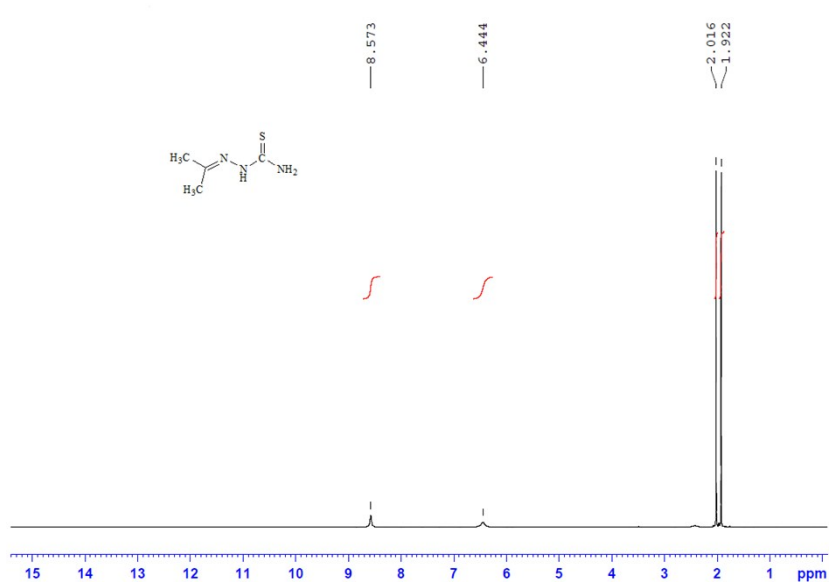


Fig. S2. ^1H NMR spectrum of [H-Ac-tsc] (HL^1)

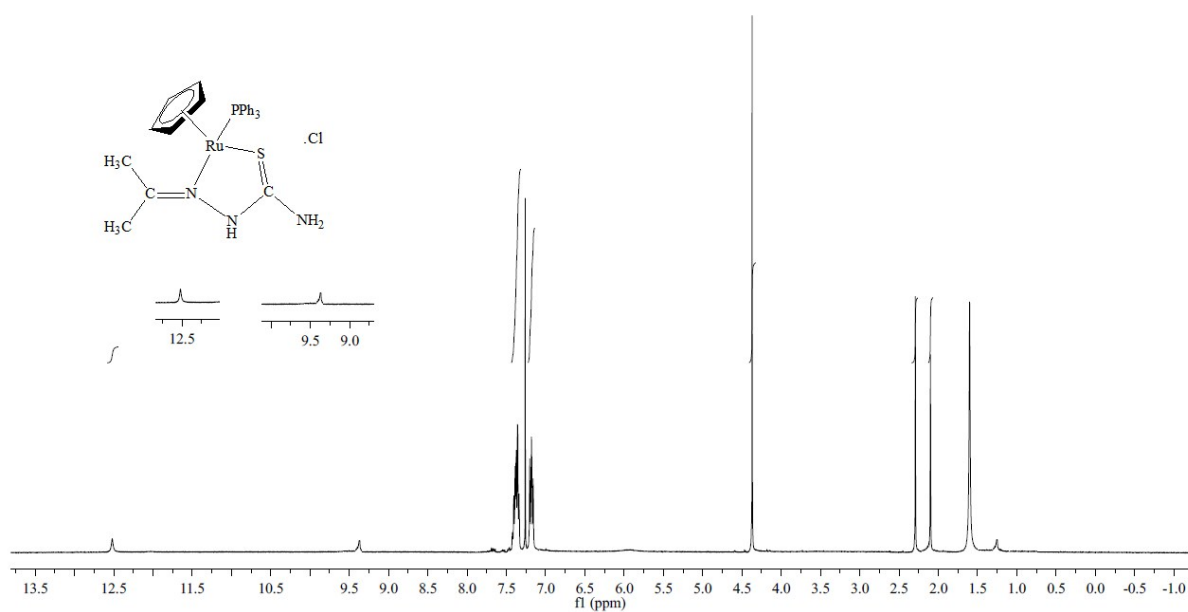


Fig. S3. ^1H NMR spectrum of $[\text{RuCp}(\text{Ac-tsc})(\text{PPh}_3)]\cdot\text{Cl}$ (1)

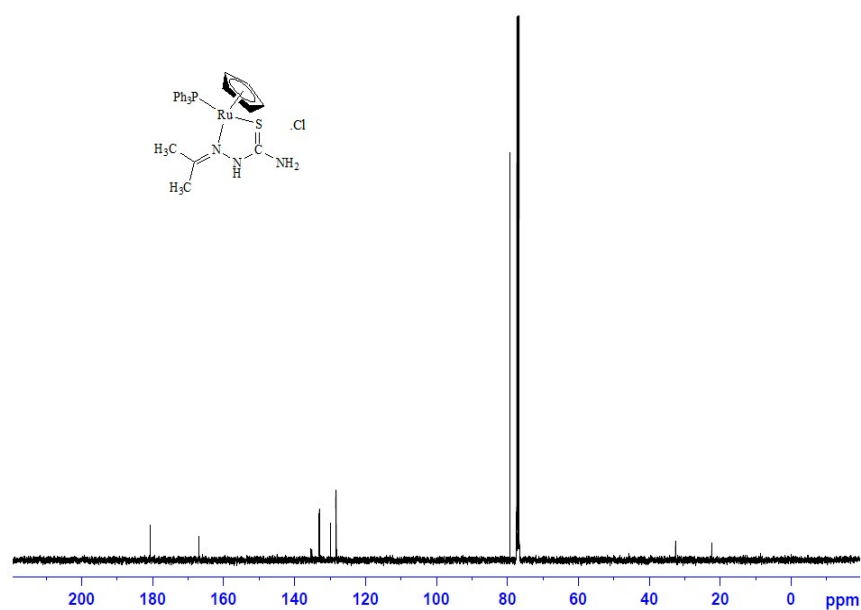
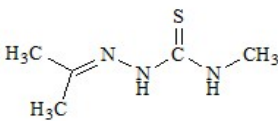


Fig. S4. ^{13}C NMR spectrum of $[\text{RuCp}(\text{Ac-tsc})(\text{PPh}_3)]\cdot\text{Cl}$ (1)



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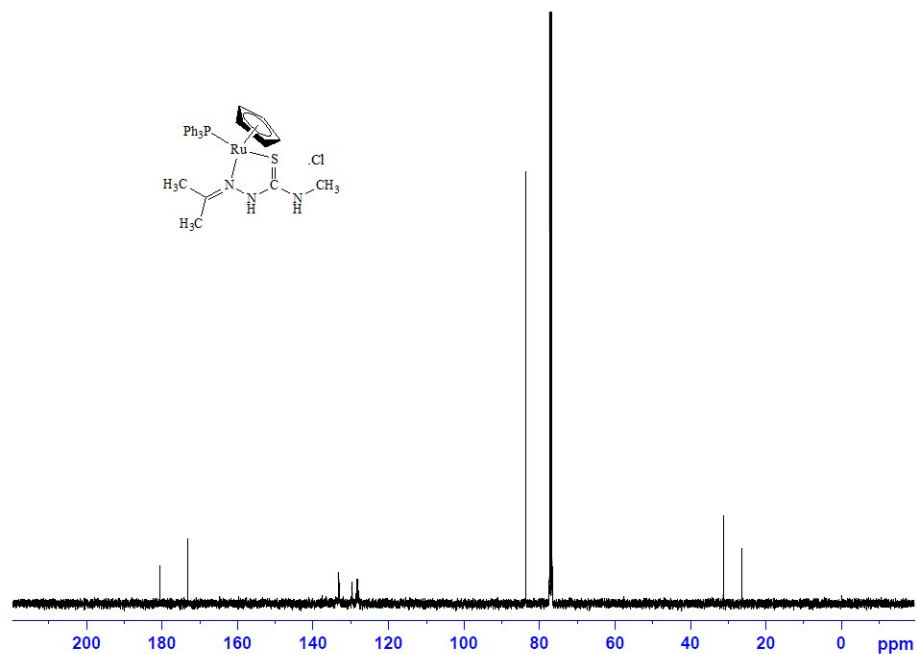


Fig. S7. ^{13}C NMR spectrum of $[\text{RuCp}(\text{Ac-mtsc})(\text{PPh}_3)]\cdot\text{Cl}$ (2)

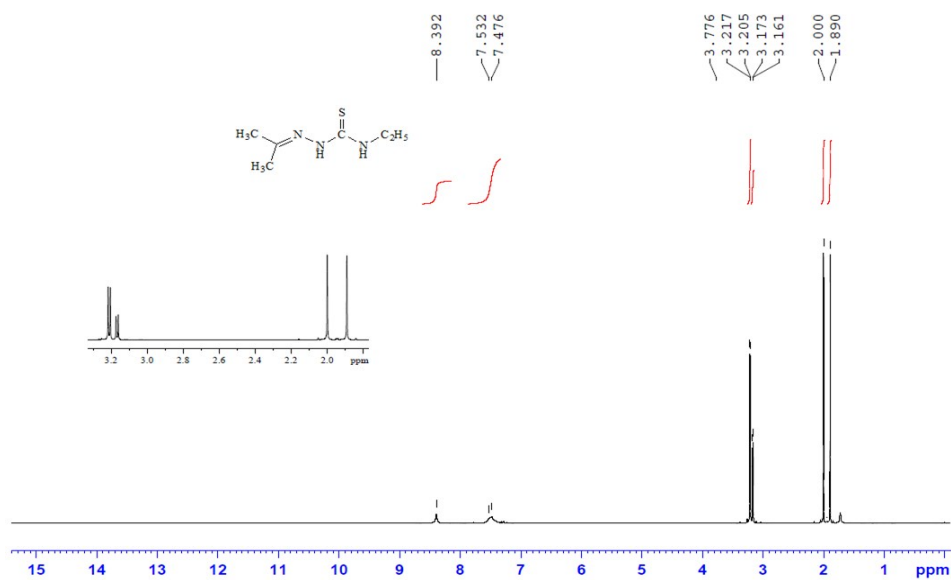


Fig. S8. ^1H NMR spectrum of $[\text{H-Ac-etsc}]$ (HL³)

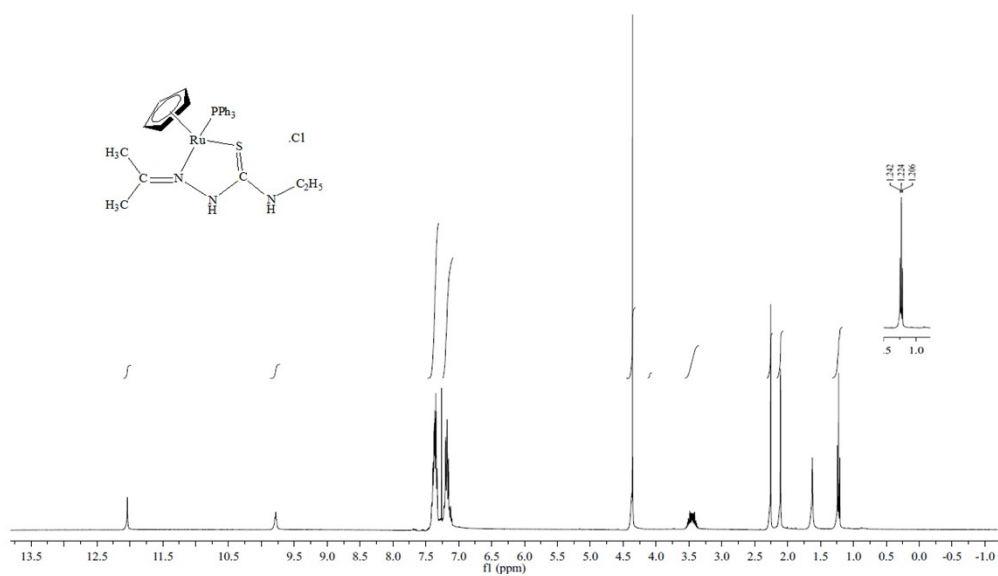


Fig. S9. ^1H NMR spectrum of $[\text{RuCp}(\text{Ac-etsc})(\text{PPh}_3)]\cdot\text{Cl}$ (3)

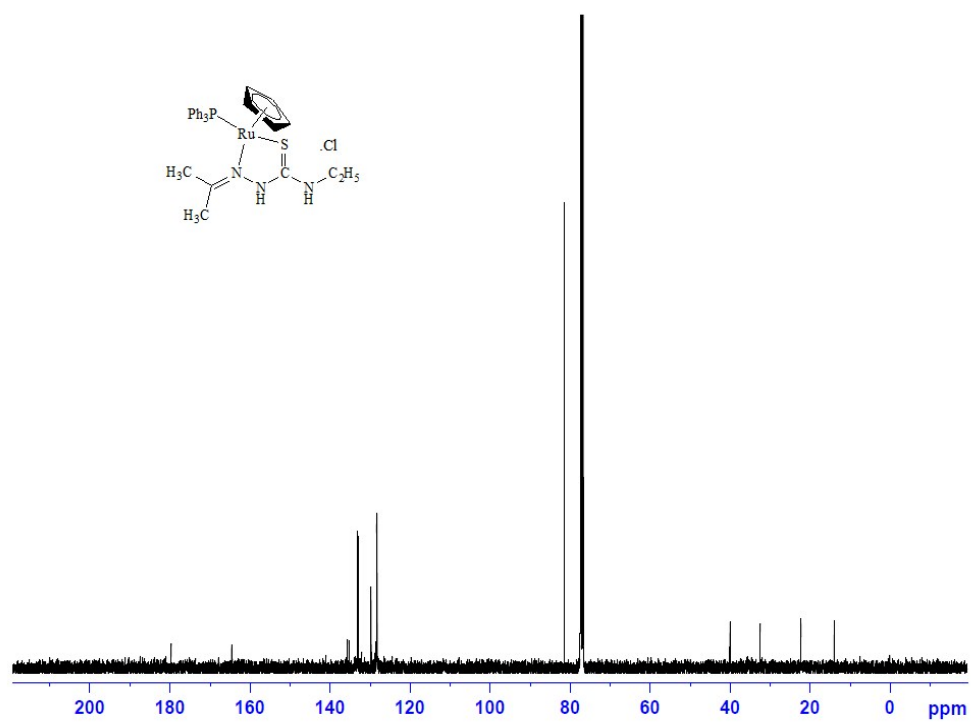


Fig. S10. ^{13}C NMR spectrum of $[\text{RuCp}(\text{Ac-etsc})(\text{PPh}_3)]\cdot\text{Cl}$ (3)

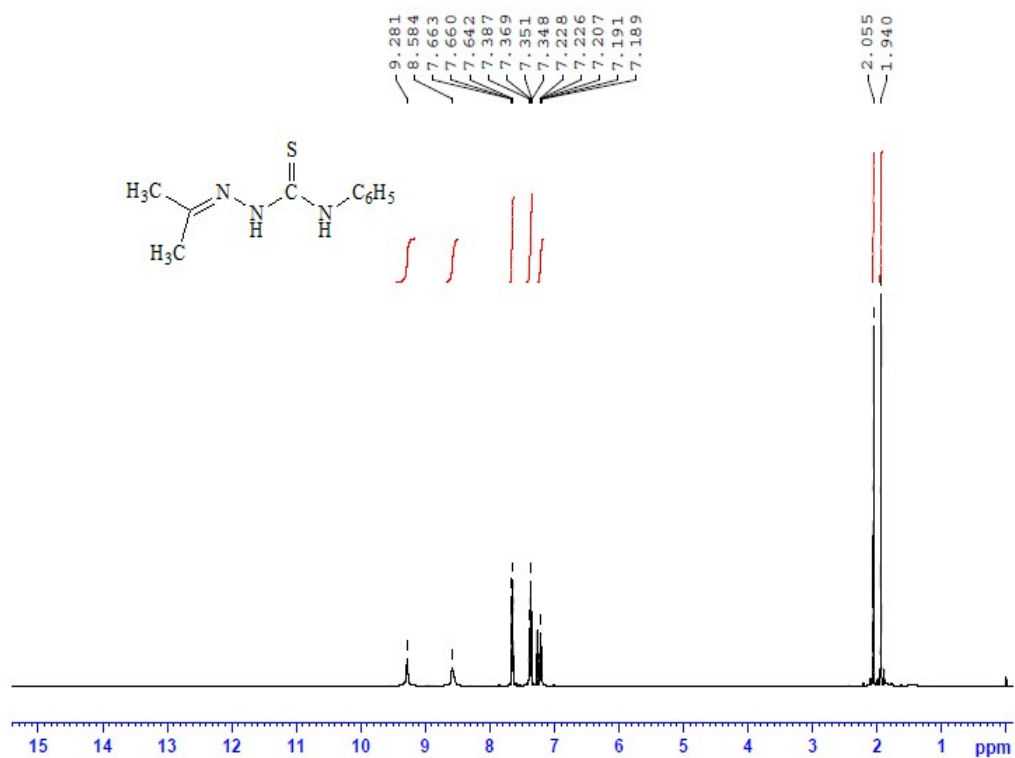


Fig. S11. ¹H NMR spectrum of [H-Ac-ptsc] (HL⁴)

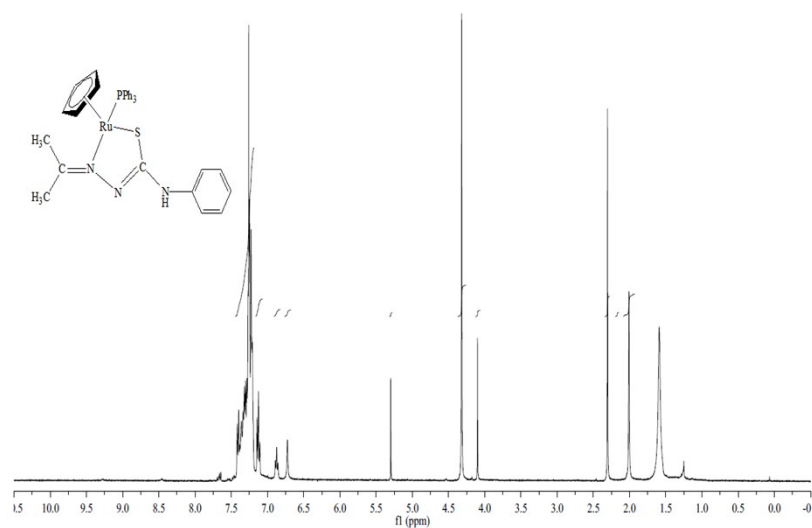


Fig. S12. ¹H NMR spectrum of [RuCp(Ac-ptsc)(PPh₃)] (4)

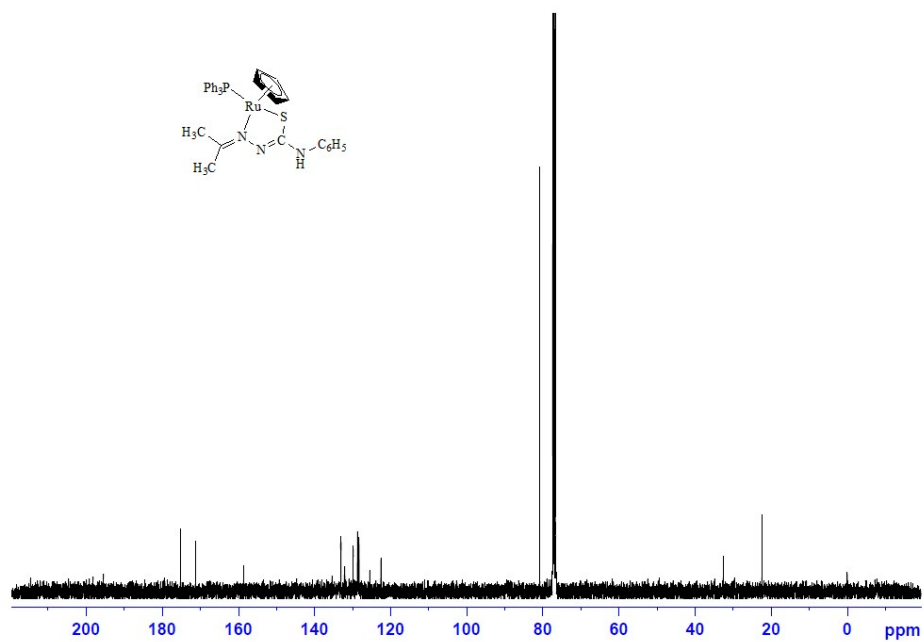


Fig. S13. ^{13}C NMR spectrum of $[\text{RuCp}(\text{Ac-ptsc})(\text{PPh}_3)]$ (4)

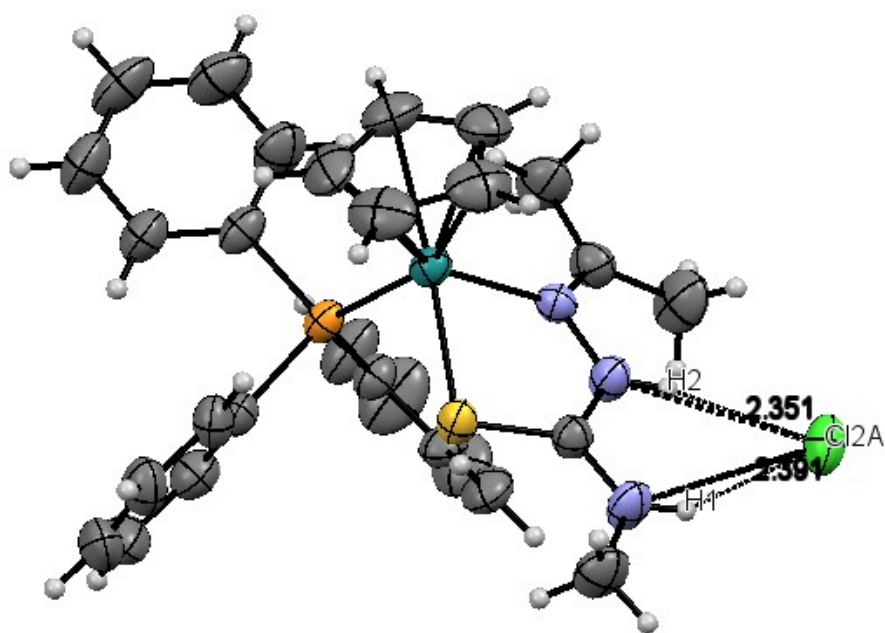


Fig. S14. Hydrogen bonding of $[\text{RuCp}(\text{Ac-mtsc})(\text{PPh}_3)] \cdot \text{Cl}$ (2)

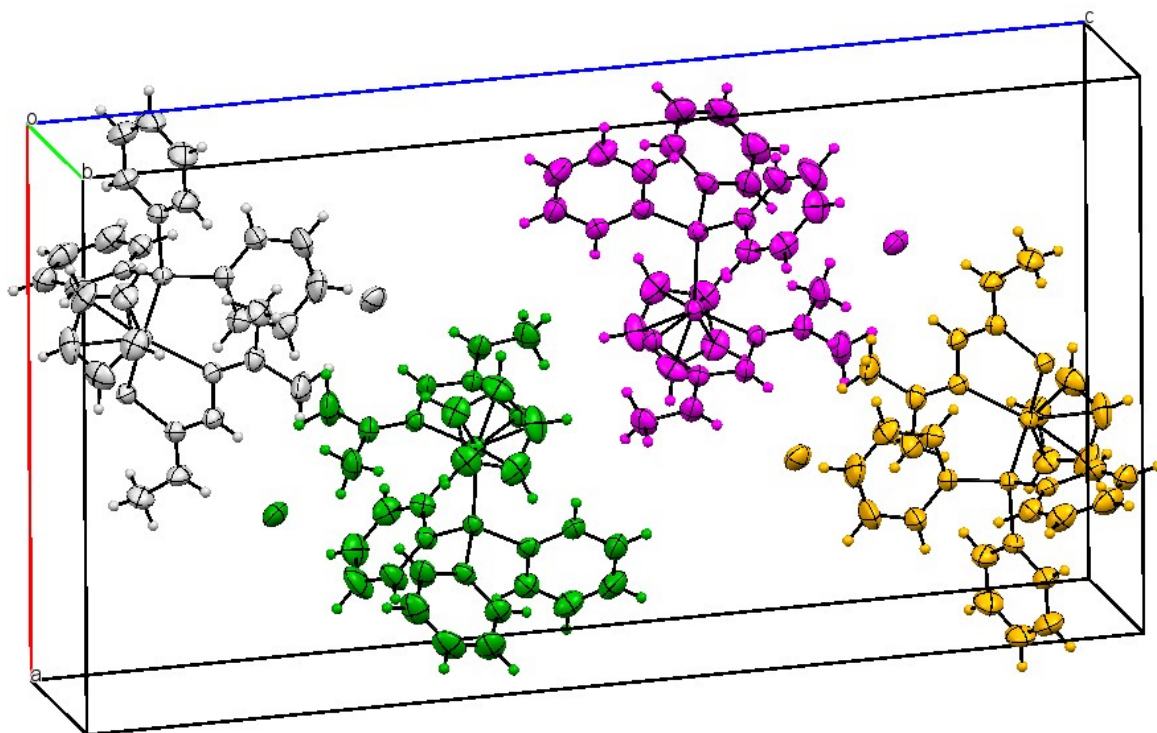


Fig. S15. Molecular packing diagram of $[\text{RuCp}(\text{Ac-mtsc})(\text{PPh}_3)]\cdot\text{Cl}$ (2)

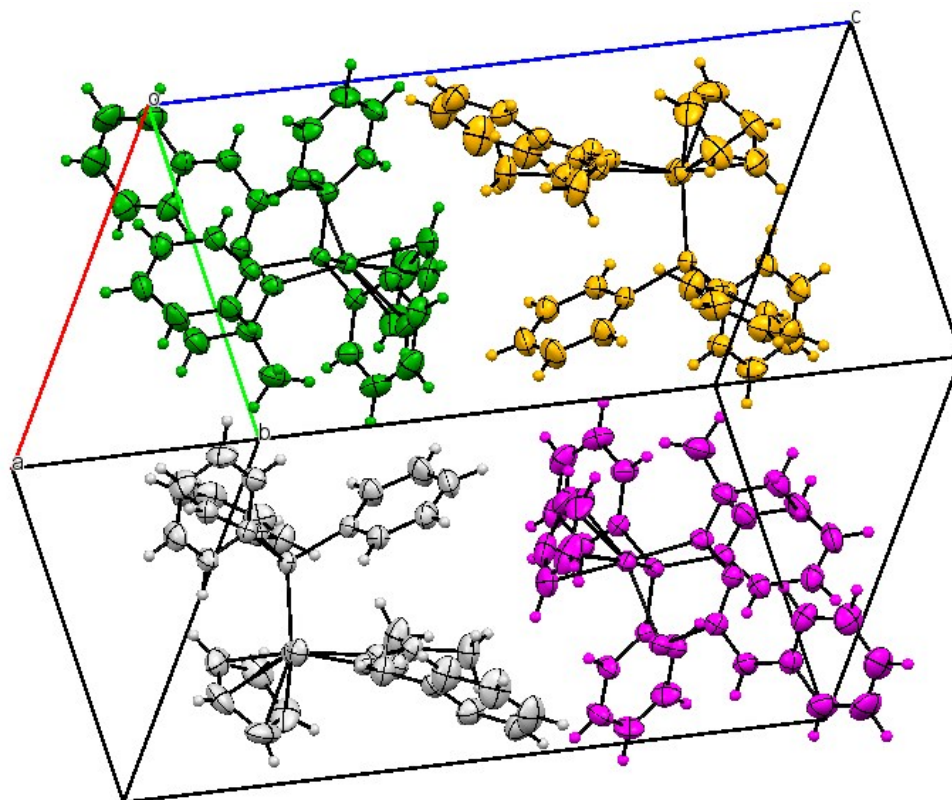
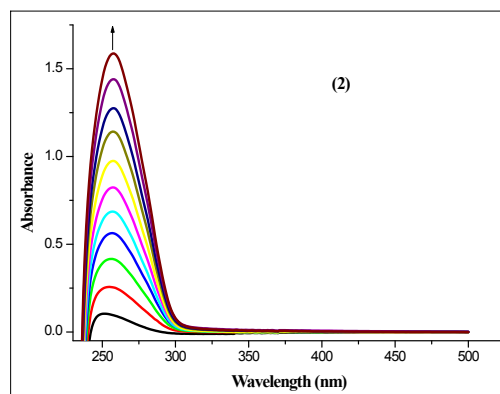
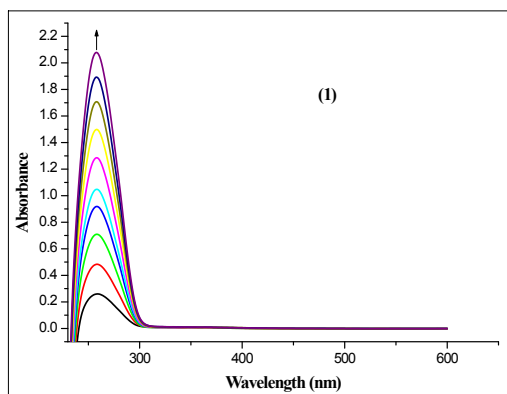
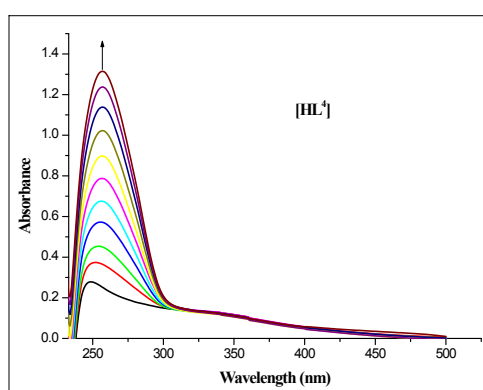
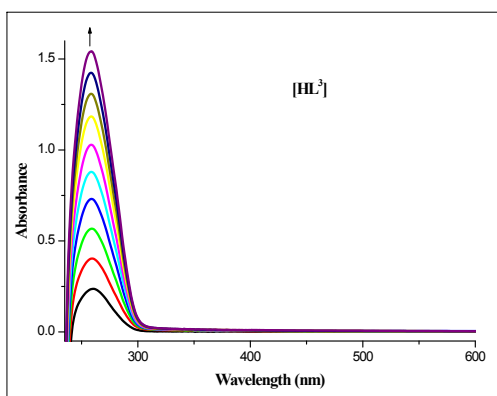
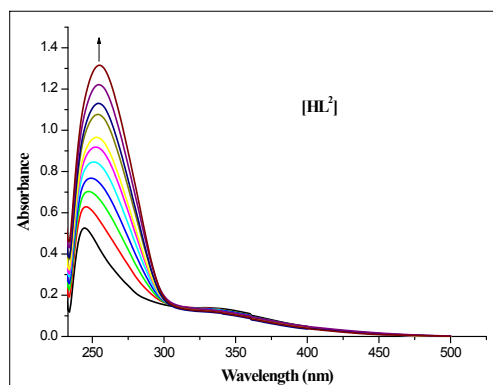
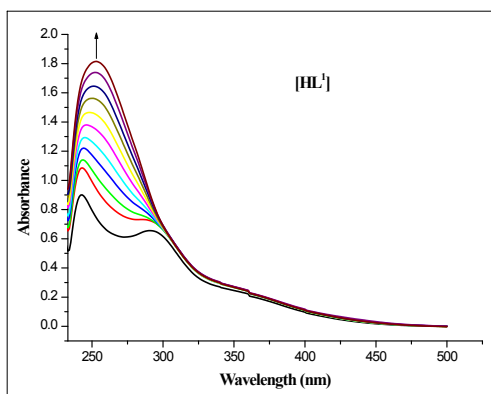


Fig. S16. Molecular packing diagram of $[\text{RuCp}(\text{Ac-ptsc})(\text{PPh}_3)]$ (4)



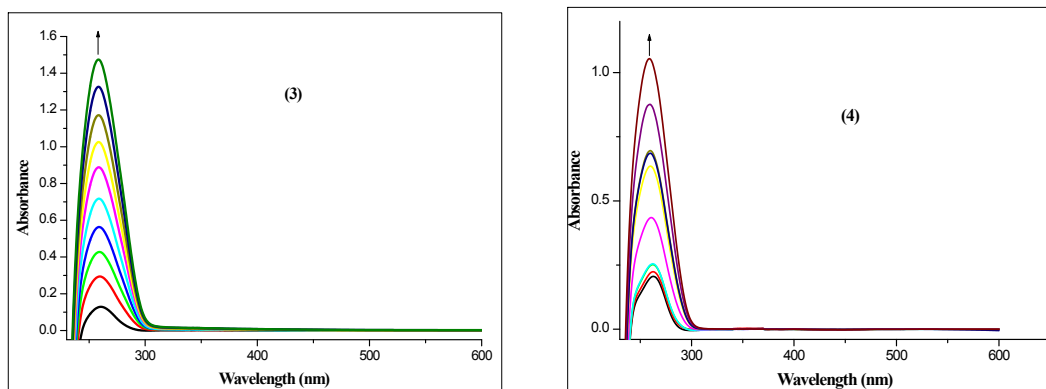


Fig. S17. Absorption titration spectra of fixed concentration (10 μ M) of ligands (HL¹-HL⁴) complexes **1-4** with increasing concentrations (0 - 50 μ M) of CT-DNA (trisHcl buffer, pH 7.2).

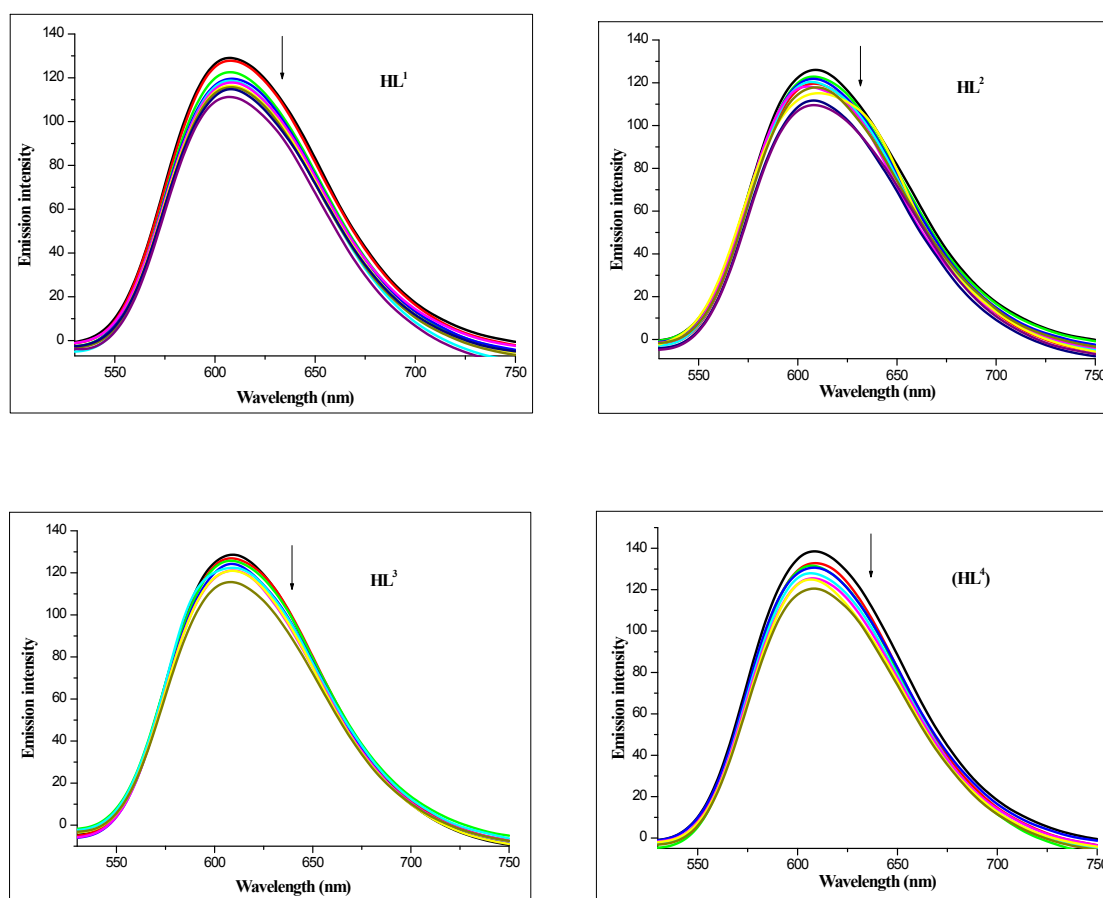


Fig. S18. The emission spectra of the DNA–EB system ($\lambda_{exc} = 515$ nm, $\lambda_{em} = 550$ – 750 nm), in the presence of ligands (**HL**¹⁻⁴). [DNA] = 10 μ M, [ligands] = 0–50 μ M, [EB] = 10 μ M. The arrow shows the emission intensity changes upon increasing ligands concentration.

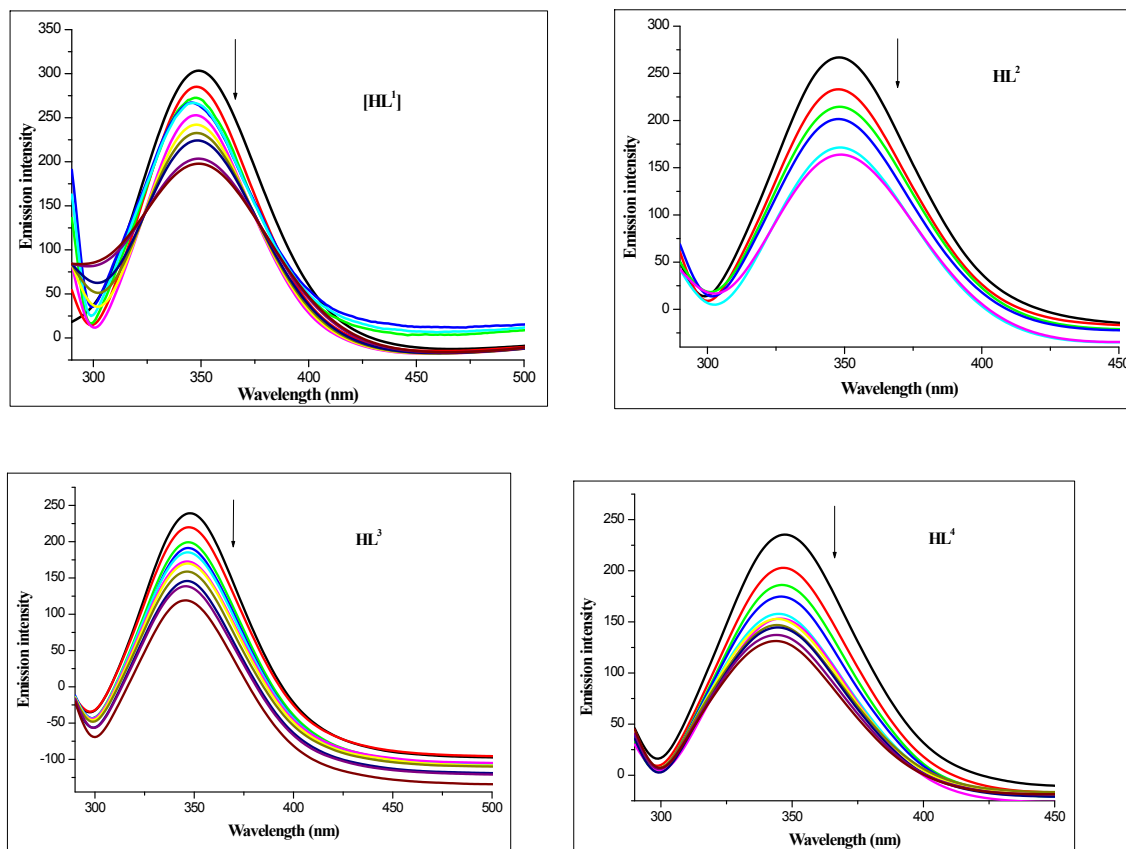


Fig. S19. The emission spectrum of BSA (10 μ M; $\lambda_{exc} = 278$ nm; $\lambda_{emi} = 347$ nm) in the presence of increasing concentration of ligands (0 – 50 μ M). The arrow shows the emission intensity changes upon increasing ligands concentration.

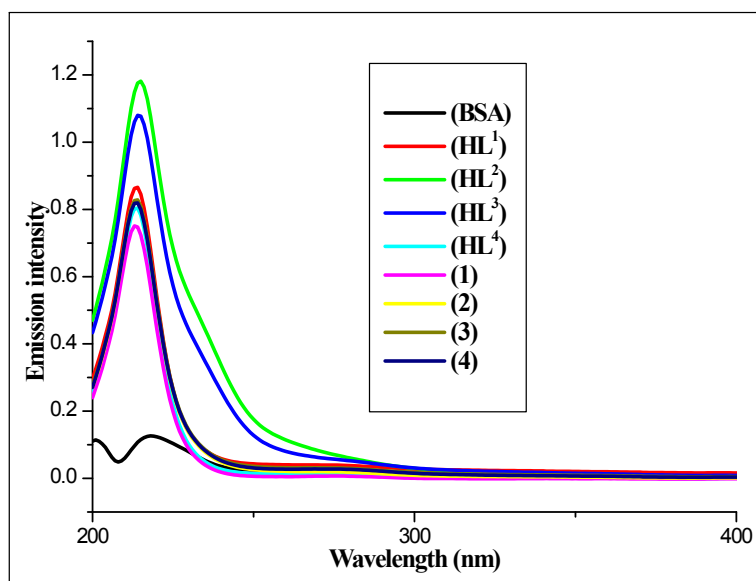


Fig. S20. Absorption titration spectra of ligands and complexes with BSA

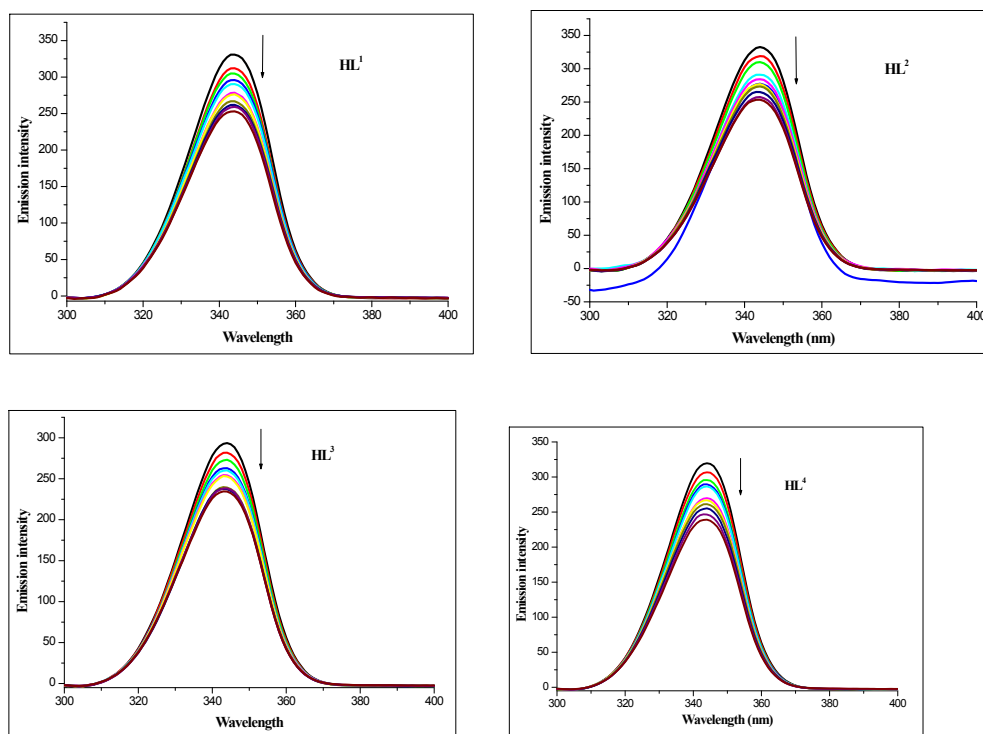


Fig. S21. Synchronous spectra of BSA (10 μM) in the presence of increasing concentration of ligands HL¹-HL⁴ for a wavelength difference of $\Delta\lambda = 60$ nm. The arrow shows the emission intensity changes upon increasing concentration of ligands.

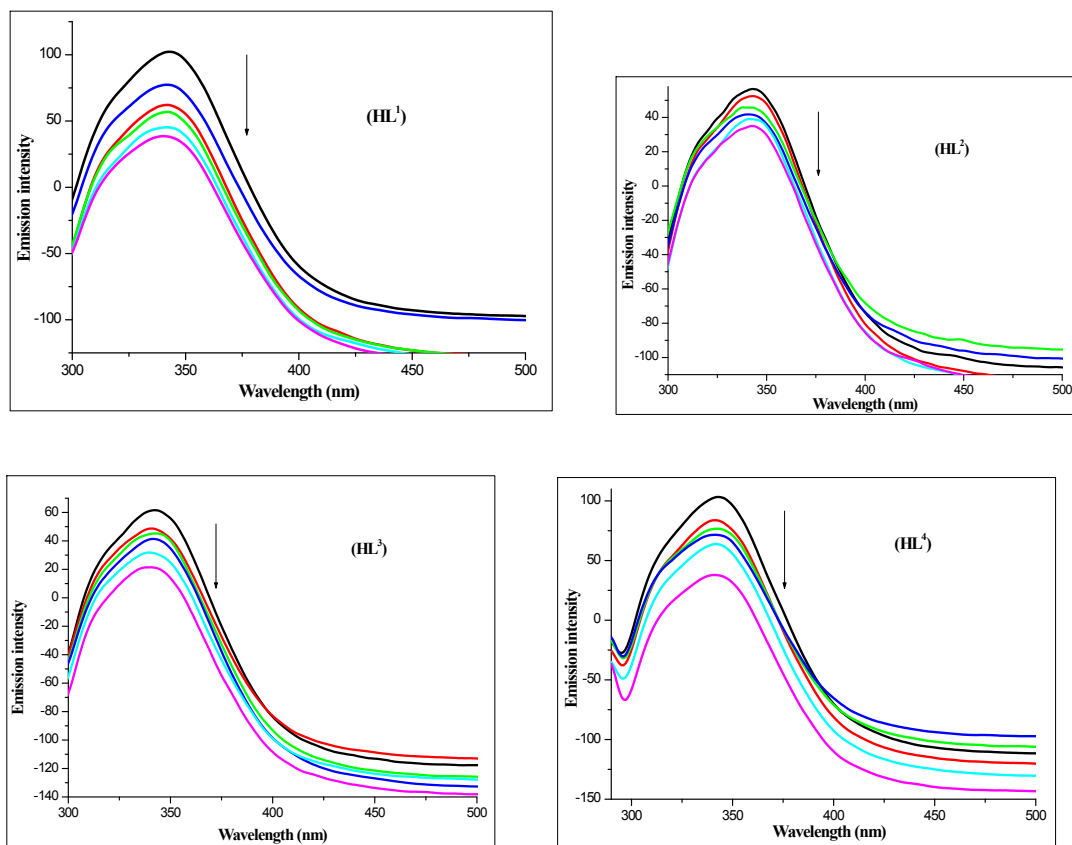


Fig. S22. The emission spectrum of HSA (10 μM ; $\lambda_{\text{exc}} = 280 \text{ nm}$; $\lambda_{\text{emi}} = 340 \text{ nm}$) in the presence of increasing concentration of ligands (0 – 50 μM). The arrow shows the emission intensity changes upon increasing ligands concentration.

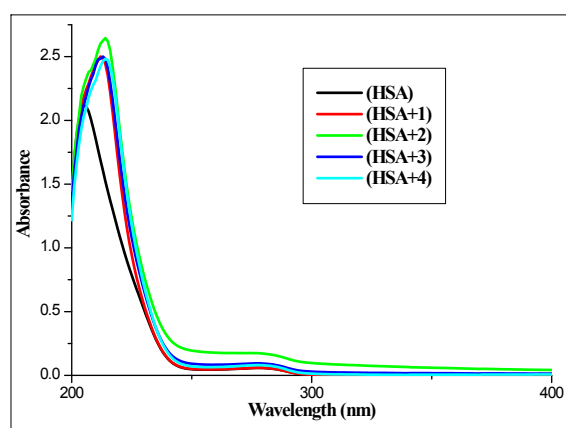


Fig. S23. Absorption titration spectra of complexes with HSA

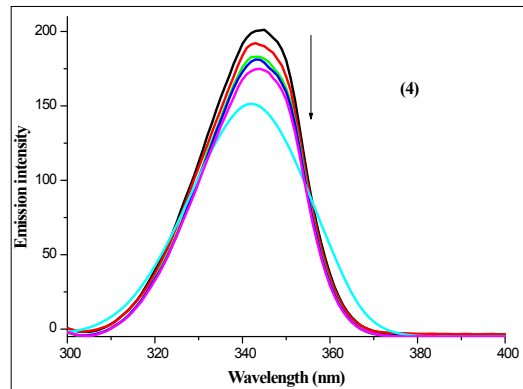
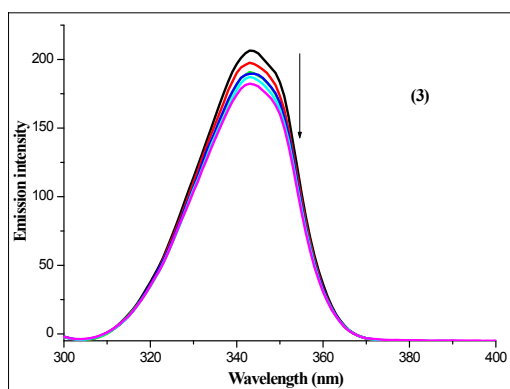
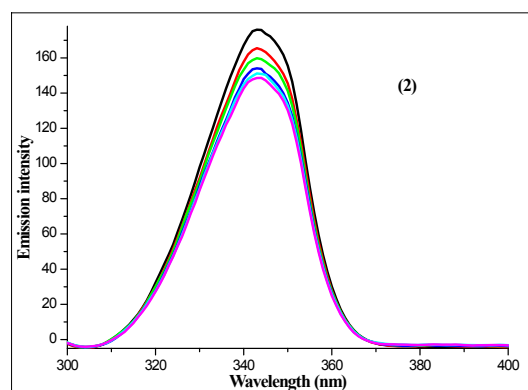
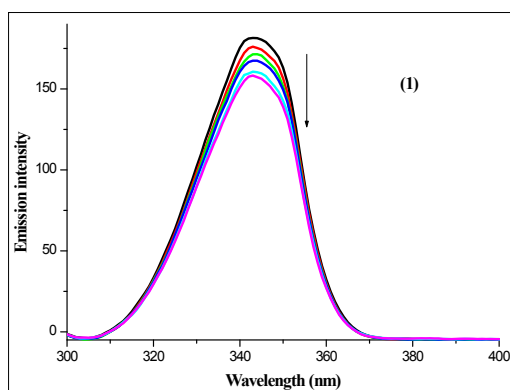
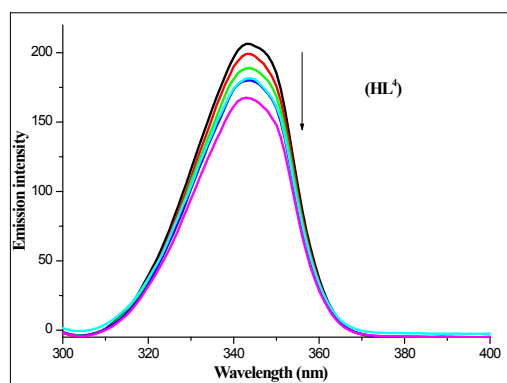
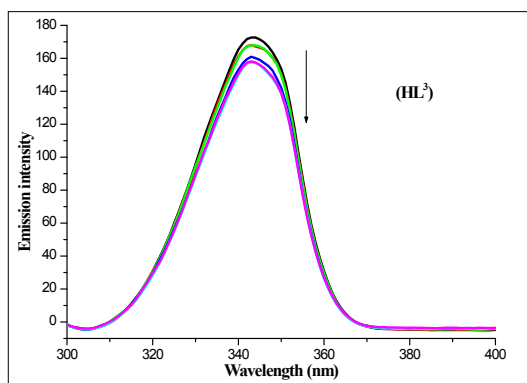
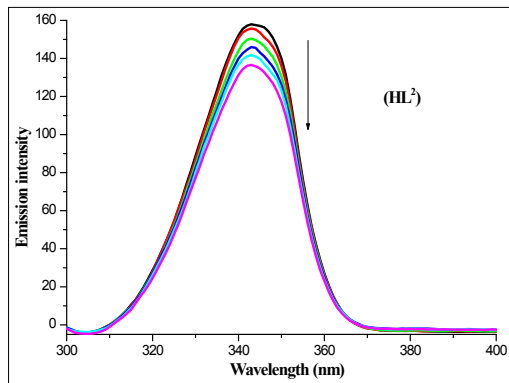
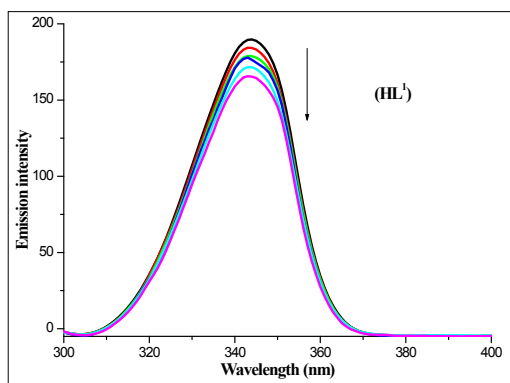


Fig. S24. Synchronous spectra of HSA (10 μ M) in the presence of increasing concentration of Ligands (**HL**¹-**HL**⁴) and complexes (**1-4**) for a wavelength difference of $\Delta\lambda = 60$ nm. The arrow shows the emission intensity changes upon increasing concentration of ligands.

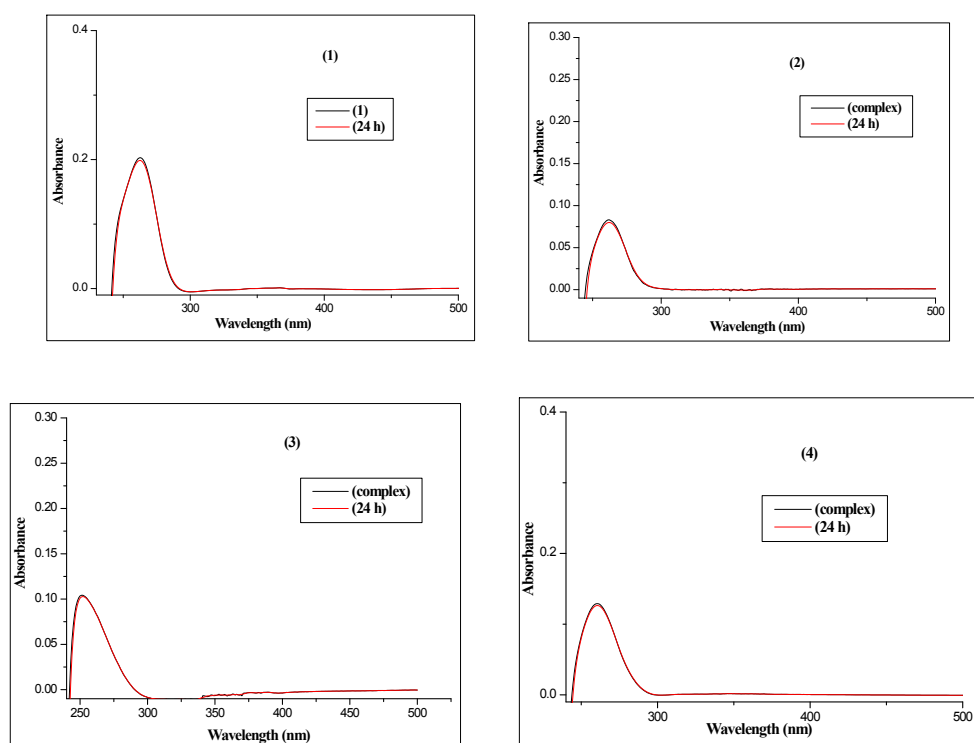


Fig. S25. Stability of the complexes (1-4) in 0.2 % DMSO

Table S1. Comparative results of DNA, BSA, HSA binding and cytotoxicity studies

	DNA Binding study	BSA Binding study	HSA Binding study	Cytotoxicity studies (μM)(IC_{50})
HL ¹	0.83×10^5	0.23×10^4	2.70×10^4	No Activity
HL ²	1.63×10^5	0.63×10^4	1.39×10^5	No Activity
HL ³	2.10×10^5	0.50×10^4	8.93×10^4	No Activity
HL ⁴	2.35×10^5	0.03×10^4	3.72×10^4	No Activity
Complex 1	3.71×10^5	3.44×10^4	1.87×10^5	27.27
Complex 2	1.96×10^6	3.97×10^6	4.27×10^6	10.67
Complex 3	9.52×10^5	1.12×10^6	6.74×10^5	11.13
Complex 4	5.36×10^5	9.32×10^4	4.49×10^5	17.85

References

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