Supplementary information

The viability of the complexes and ligand was analysed in TZM-bl cells and PBMCs (Figure S1 and S2). The ligand did not have viability values below 50% and was only analysed in TZM-bl cells, with this the CC_{50} value of the ligand is >200 µM.

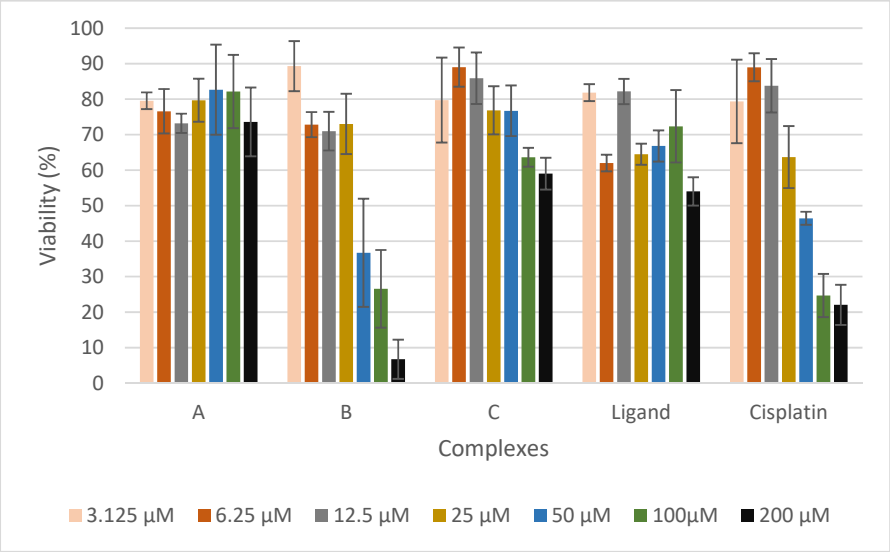

Figure S1: The viability of the TZM-bl cells in the presence of the metal-based complexes, the ligand and Cisplatin as the positive control. N-aryl-1H-1,2,3- triazole-based cyclometalated ruthenium-(II) complex (***A***), N-aryl-1H-1,2,3- triazole-based cyclometalated osmium-(II) complex (***C***) and the ligand were found to be non-toxic as the viability did not go below 50% at any of the analysed concentrations. N-aryl-1H-1,2,3- triazole-based cyclometalated iridium-(III) complex (***B***) was found to have a CC_{50} value of 36.13 ± 2.99 µM and Cisplatin was found to have a value of 43.52 ± 3.45 µM. The inhibition values were reported as the mean ± SEM for n=3.

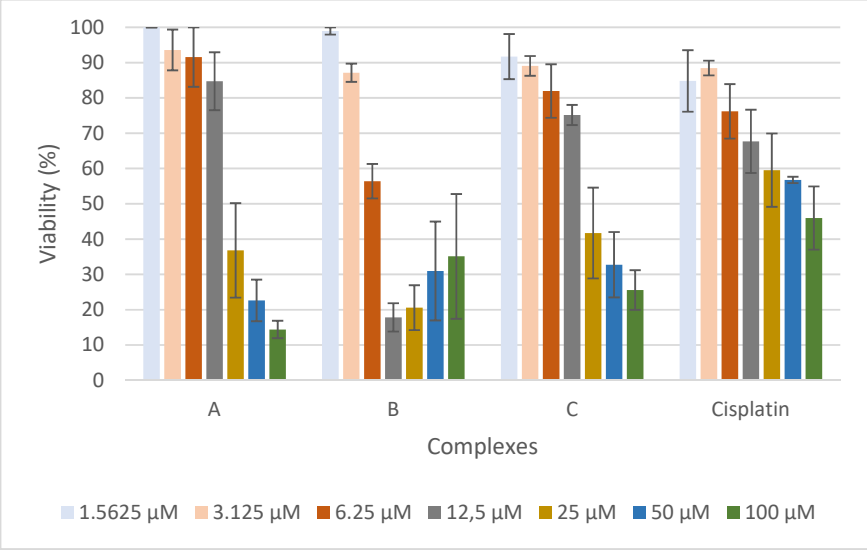

Figure S2: The viability of PBMCs in the presence of the metal-based complexes and Cisplatin as the positive control. All three the metal-based complexes were found to be more toxic than in TZM-bl cells. N-aryl-1H-1,2,3- triazole-based cyclometalated ruthenium-(II) complex (***A***) had a CC_{50} value of 24.36 ± 5.98 µM. N-aryl-1H-1,2,3- triazole-based cyclometalated Iridium-(III) complex (***B***) had a CC_{50} value of 13.09 ± 3.18 µM. N-aryl-1H-1,2,3- triazole-based cyclometalated osmium-(II) complex (***C***) had a CC_{50} value of 29.16 ± 8.03 µM. The control Cisplatin had a CC_{50} value of 49.09 ± 3.26 µM. The inhibition values are reported as mean± SEM for n=3.

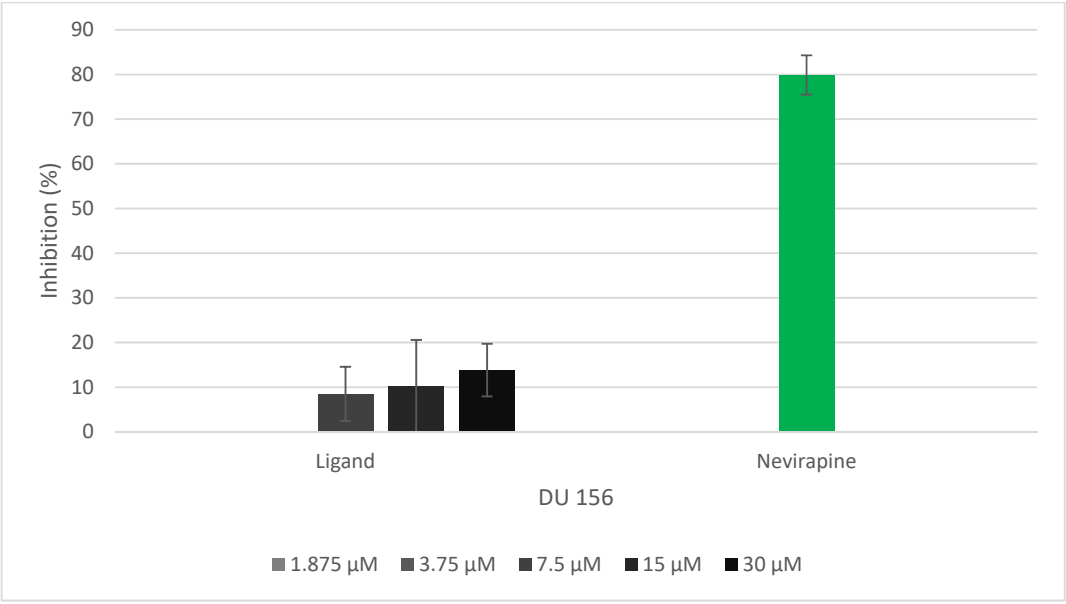

Figure S3: The percentage inhibition of the pseudo-virus Du 156. Inhibition of Du 156 following the treatment of the ligand at similar concentrations to the complexes. The inhibition values reported as mean ± SEM for n = 3.

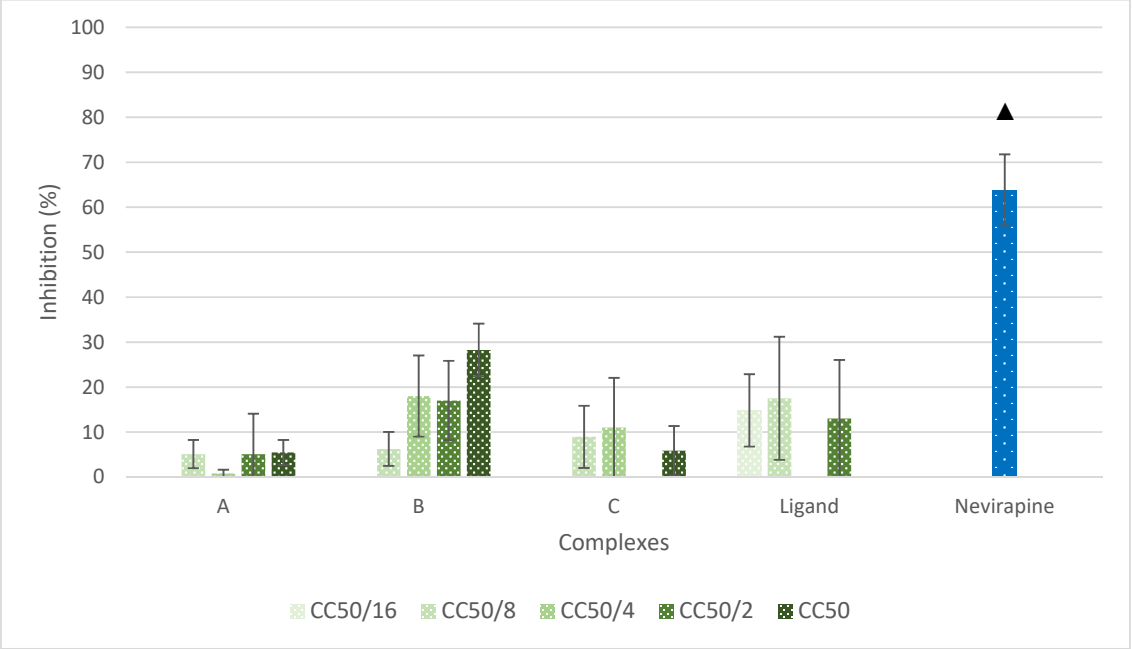

Figure S4: Luciferase inhibition of the complexes and the ligand. Luciferase inhibition of the complexes as well as the ligand with Nevirapine as control (▲ pre-treated to prevent production of luciferase). The same concentrations were analysed as with the luciferase reporter gene assay, starting with a serial dilution from the CC_{50} values of the PBMCs. The inhibition of luciferase was found not to be statistically significant when comparing the luciferase produced in pseudo-virus only infected cells to the pseudo-virus cells lysed and treated with the complexes. The inhibition values reported as mean ± SEM for n = 3.