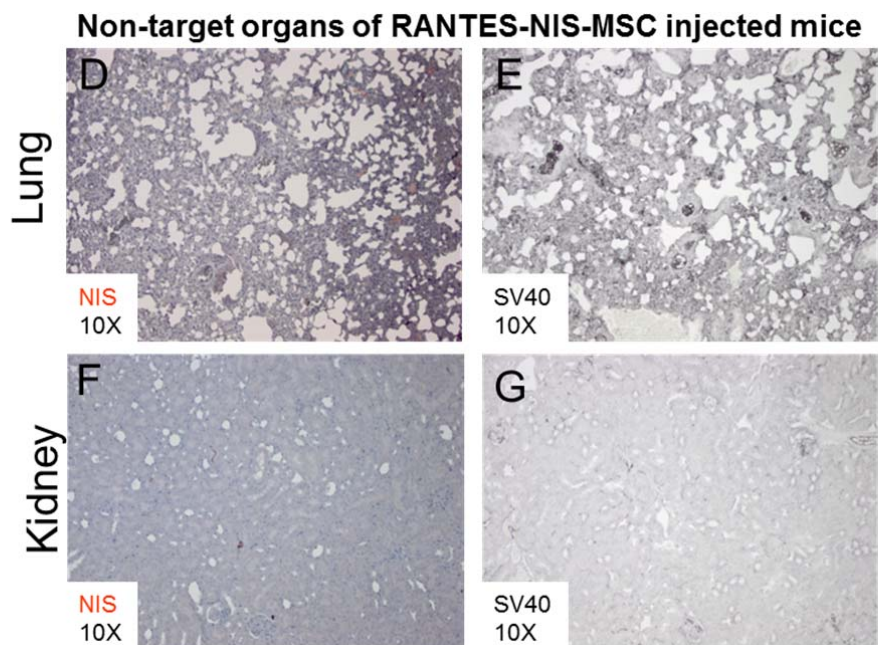
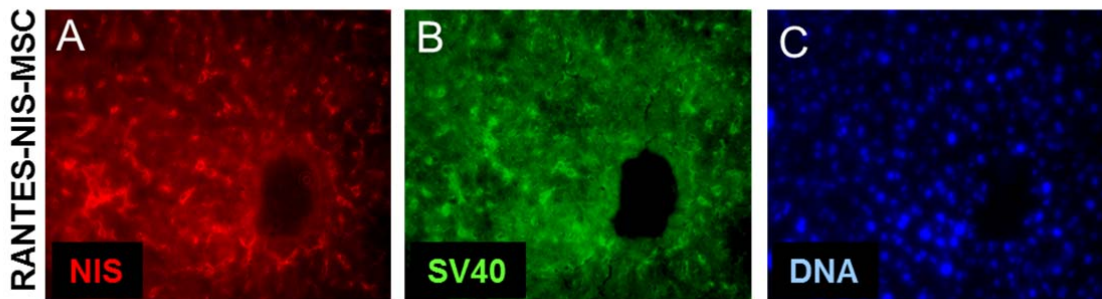
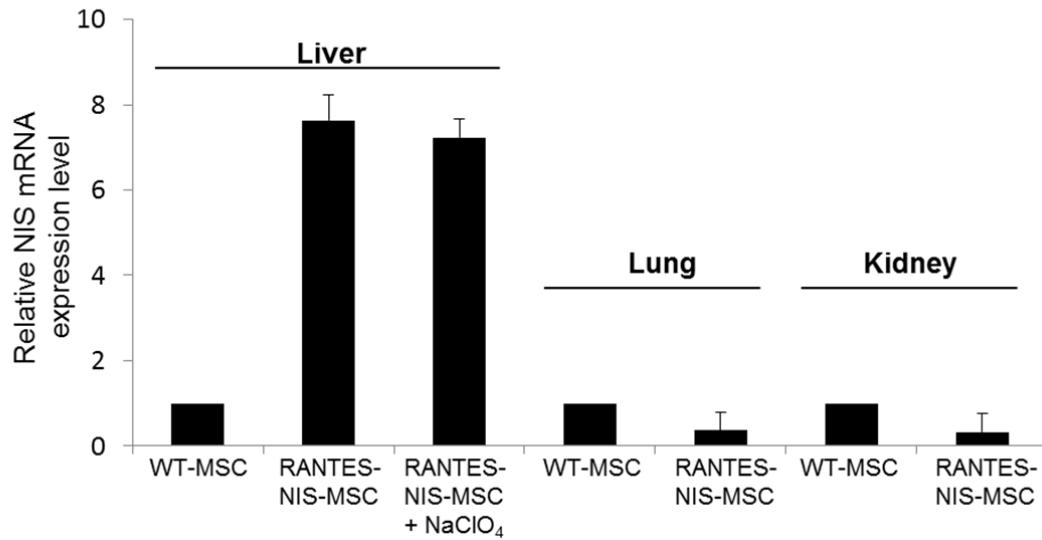


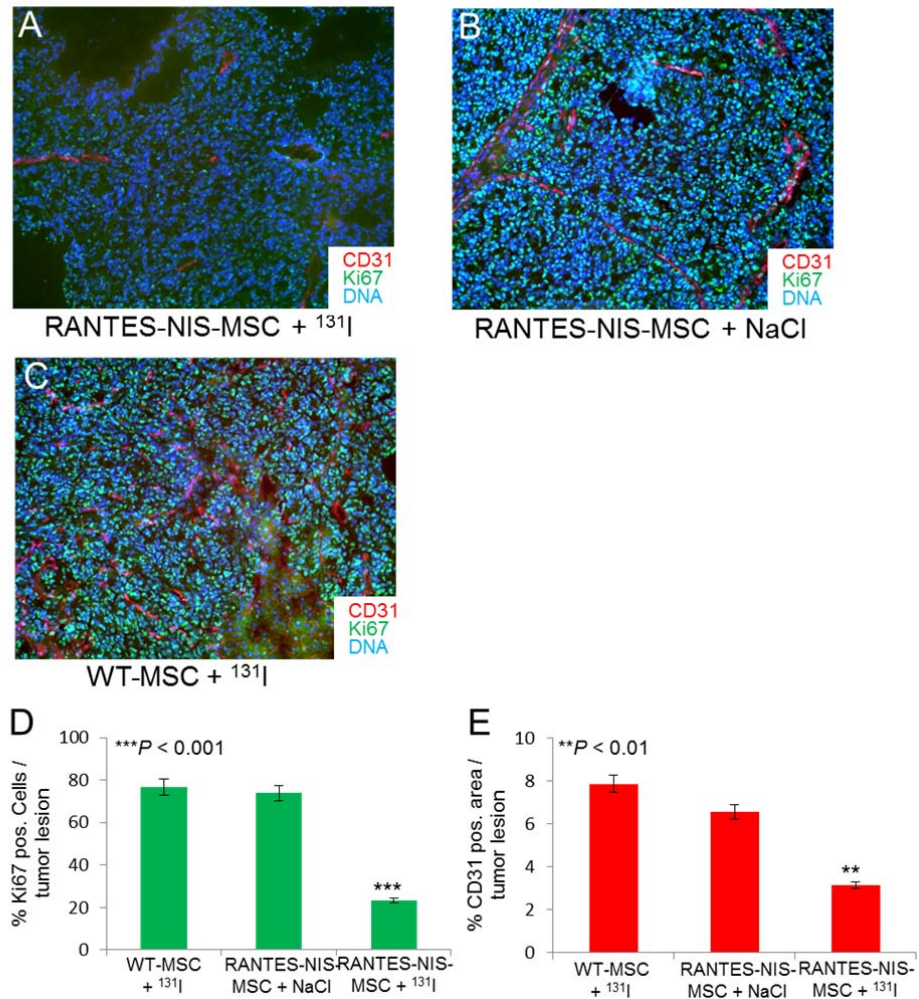
Supplemental Figure 1: Iodide uptake activity measured by ^{123}I -scintigraphy resulted in a maximum uptake of 12.1 ± 2.6 % ID/g with a biological half-life of 2.9 hours and a tumor absorbed dose of 63.2 mGy/MBq.



Supplemental Figure 2: Immunofluorescence analyses confirmed the co-localisation of RANTES promoter-mediated NIS expression with MSC-specific SV40 large T Ag-specific staining (A – C). Non-target organs including lung and kidney were negative for NIS-specific immunostaining (D, F) as well as MSC-specific SV40 large T Ag-specific staining (E, G).



Supplemental Figure 3: Quantitative real time PCR revealed only background levels of NIS mRNA expression in liver metastases of mice injected with WT-MSC, whereas significant levels of NIS mRNA expression were detected in the livers of mice harbouring colon cancer liver metastases after application of RANTES-NIS-MSC injections (7.6-fold) and in mice additionally treated with the competitive NIS inhibitor perchlorate (NaClO₄) (7.4-fold). In non-target organs including lung and kidney no NIS mRNA expression was seen after injection of RANTES-NIS-MSC or WT-MSC.



Supplemental Figure 4: Immunofluorescence analysis of liver metastases using a Ki67-specific antibody (green, labelling proliferating cells) and an antibody against CD31 (red, labelling blood vessels) showed striking difference in tumor cell proliferation and blood vessel density in metastases of mice treated with RANTES-NIS-MSCs and ¹³¹I (A), which was significantly reduced (Ki67: 23.3 % ± 2.5%; CD31: 3.1% ± 0.2% (D, E)) as compared to the control groups of mice treated with RANTES-NIS-MSC and saline (B) (Ki67: 73.8% ± 6.9%, CD31: 6.6% ± 0.7% (D, E)) or WT-MSC and ¹³¹I (C) (Ki67: 76.5% ± 6.7%, CD31: 7.8% ± 0.8% (D, E)). Slides were counterstained with Hoechst nuclear stain. Magnification x200.