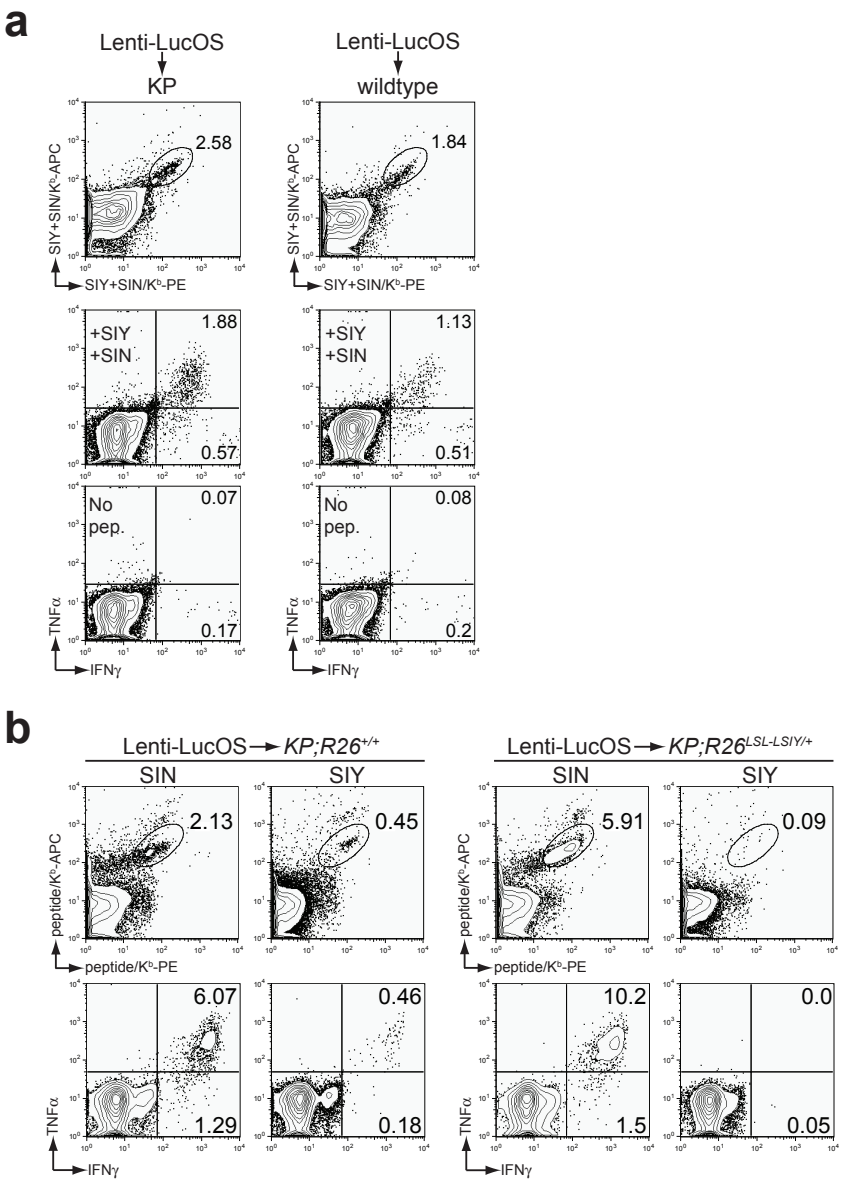
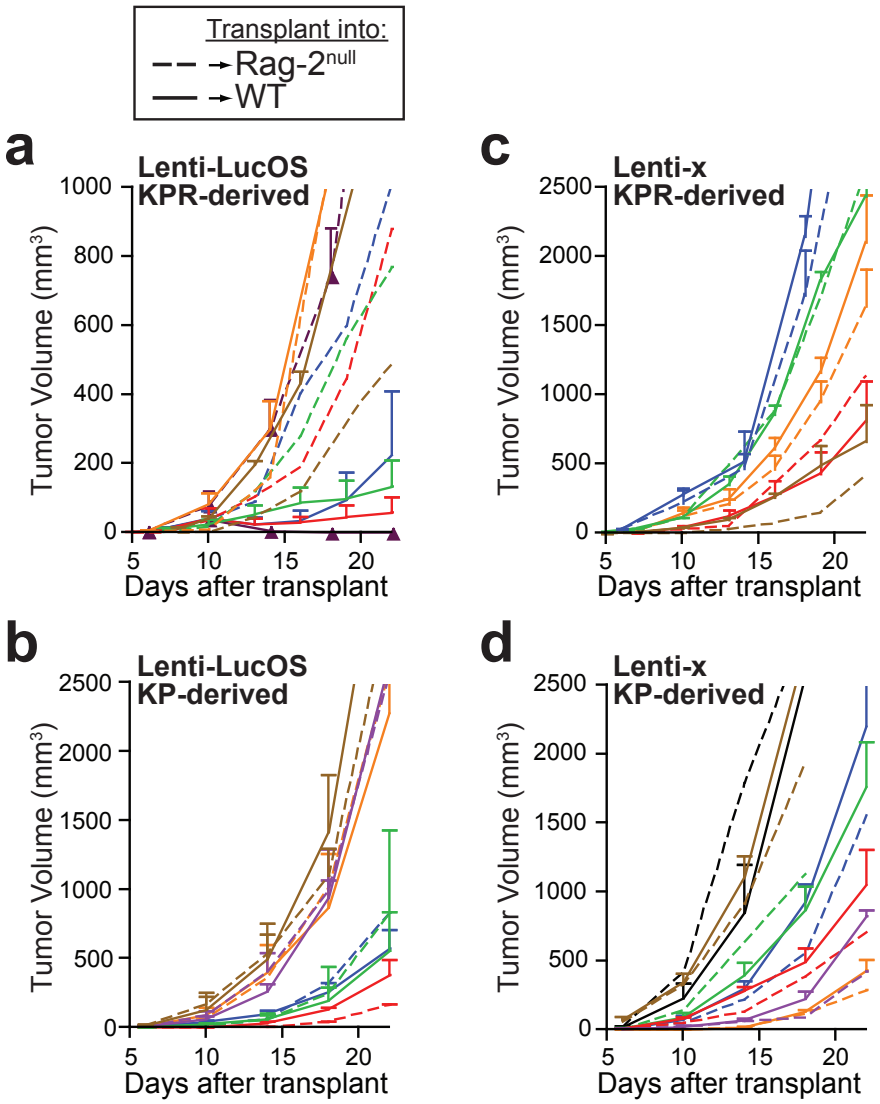
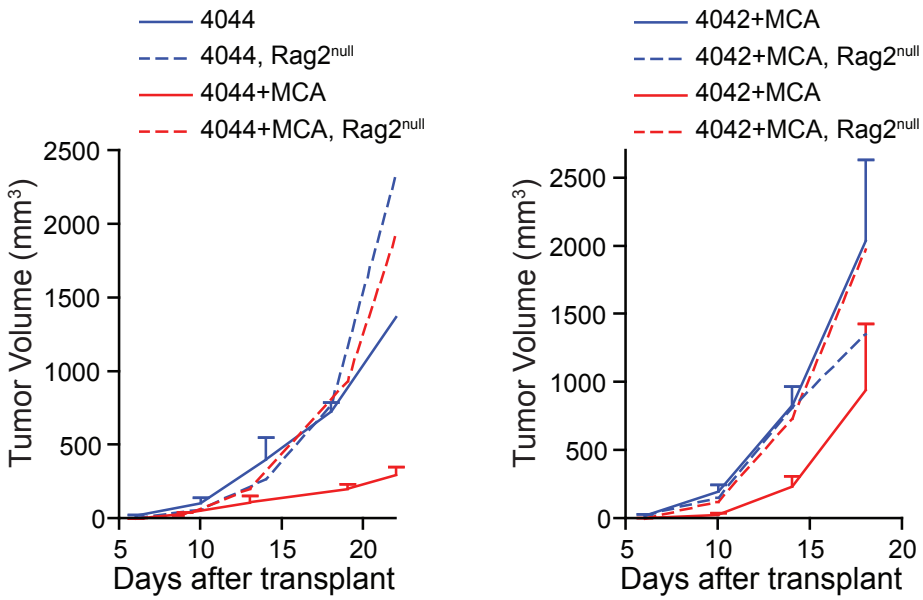


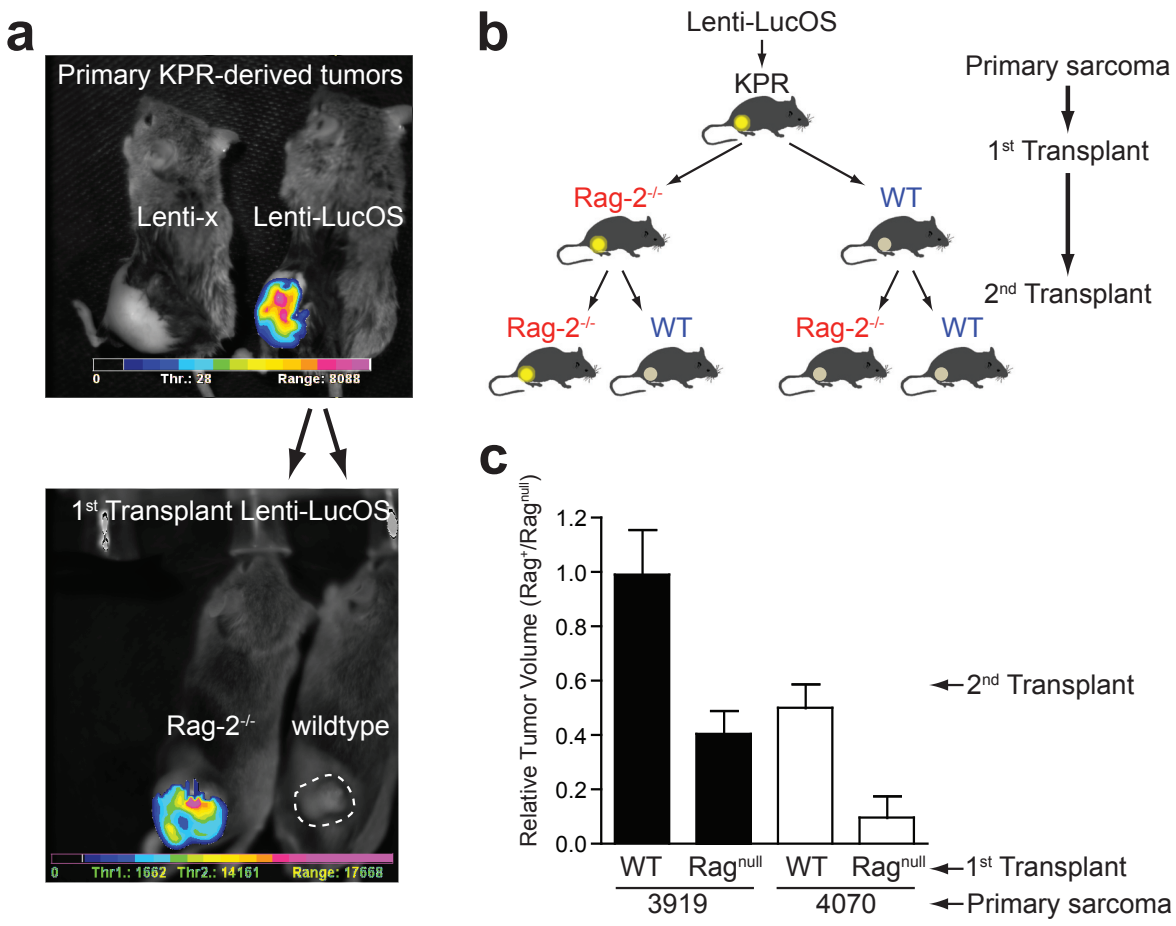
Supplementary Figure 1. T cell responses are specific to exogenously introduced tumour-specific antigens expressed in lentiviruses and tumours. (**a**) KP (left) or wild-type mice (right) were infected intramuscularly with Lenti-LucOS and analyzed five weeks later (no tumours were apparent). *Top:* The percent of $CD8^+$ cells specific for SIY- and SIN-loaded K^b dimers in the spleen. Plots are gated on PI-negative, $CD8^+$ cells. *Bottom:* IFN-γ and TNF-α cytokine production in CD8-gated T cells from the spleens of mice analyzed above after restimulation *in vitro* with both SIY and SIN peptides or not restimulated. Representative of the analysis of three mice per group.
(**b**) Comparison of KP (left) or KPL-SIY mice (right) infected intramuscularly with Lenti-LucOS shows a lack of SIY-specific T cells in mice with the *R26*$^{LSL\text{-}LSIY}$ allele. Analysis performed as in (a) except cells were stained separately with SIY or SIN-loaded K^b dimers and restimulated separately with SIY or SIN. Representative of the analysis of at least 2 mice per group.



Supplementary Figure 2. Cancer immunoediting phenotypes require the presence of potent T cell antigens. Comparison of transplanted tumour growth of Lenti-LucOS-induced sarcomas generated in KPR (**a**) or KP (**b**) mice and Lenti-x-induced sarcomas generated in KPR (**c**) or KP (**d**) mice. Results plotted in (a-d) represent the mean tumour volume ± s.e.m. in 3-5 wild-type (solid lines) or 1-2 Rag-2$^{-/-}$ (dashed lines) recipients. Different colored lines denote different primary sarcomas. Note: one KPR-derived, Lenti-LucOS sarcoma exhibited delayed growth in wild-type mice but was not shown in this graph due to its rapid growth beyond the scale of the other six tumours depicted.



Supplementary Figure 3. MCA treatment *in vitro* may enhance the immunogenicity of Lenti-x-induced sarcomas. Examples from two Lenti-x-induced sarcoma cell lines treated with 40 μg/ml of MCA for six days before single clones were picked and grown by limiting-dilution. Only two clones of 20 total MCA-treated clones that were subsequently transplanted showed a modest increase in immunogenicity compared to the parent (4044, left) or other MCA-treated clones (4042, right). However, because it was such a rare occurrence to find clones that had increased immunogenicity after treatment with MCA, it is possible that these clones represent variants already present within the primary tumour cell population that were naturally more immunogenic, and their immunogenicity may not be a direct effect of the MCA treatment.



Supplementary Figure 4. Reduced antigen expression and immunogenicity of KPR-derived tumours passaged through immune-competent mice. (**a**) Antigen expression in primary Lenti-LucOS-induced tumours derived in KPR mice was lost after transplantation into wild-type mice but not *Rag-2*$^{-/-}$ mice. Luciferase-negative tumour in wild-type recipient is outlined. Results are representative of transplantation of four different Lenti-LucOS-induced sarcomas generated in KPR mice. (**b**) Schematic depiction of primary sarcoma induction and subsequent transplantations as performed in (a) and (c). Antigen expressing tumours (luciferase positive) are yellow whereas antigen-loss tumours are grey. (**c**) Comparison of the relative tumour growth (measured as the mean final tumour volume in wild-type/Rag-2^{+} versus Rag-2$^{-/-}$ mice ± s.e.m.) of Lenti-LucOS-induced sarcomas derived in KPR mice previously passaged through wild-type (WT) or Rag-2$^{-/-}$ (Rag^{null}) mice. Note that upon the 2nd transplantation, tumours previously passaged through wild-type mice grew more similarly in wild-type and Rag-2$^{-/-}$ mice than tumours previously passaged through Rag-2$^{-/-}$ mice, indicating a reduction in immunogenicity that coincides with a loss in tumour antigen expression (as shown in a).