

Supplemental Figure S6: TR-APC induction results in inflammatory rewiring of the tumor

microenvironment. A, UMAP projection of aggregated scRNA-seq from dissociated tumors 5 and 10 days post injection and left untreated or TR-APC induced and colored by Genome Instability Score as a continuous variable. B, UMAP projection of aggregated scRNA-seq from dissociated tumors 5 and 10 days post injection and left untreated or TR-APC induced and colored by cell origin as informed by the Genome Instability Score and used for subsequent analyses of normal immune cells and tumor-derived cells. C, UMAP projections of scRNA-seq from indicated treatment conditions and colored according to SingleR cell annotations. **D**, UMAP projections of ProjecTIL scRNA-seq analysis of tumor infiltrating T cell phenotypes from indicated treatment conditions. Contour plots display individual T cells from the indicated treatments overlayed on the ProjecTIL-constructed UMAP of T cell phenotypic space. E, Relative frequencies of observed T cell phenotypes in the indicated treatment conditions as annotated by ProjecTIL analysis of scRNA-seq data. F, Gene modules scores of myeloid activity gene sets among normal myeloid immune infiltrate of the indicated treatment conditions. G, Relative frequency of indicated SingleR-annotated cell populations within the tumor-cell derived compartment of the indicated treatment conditions. H Kaplan-Meier survival curve of mice depleted of indicated immune population. Naïve BALB/c were mice were depleted of granulocytes (Ly6G), macrophages (F4/80), NK cells (CD122), or B cells (CD20) via I.P. antibody injection as in Figure 4A. Subsequently, RAW-112 cells were engrafted I.V. and TR-APCs were induced in vivo by exposure to doxycycline chow. n=5-10 mice/group.