Supplemental Figure Legends

Supplemental Figure S1. *Trp53* inactivation in hosts results in an augmented immunotolerant tumor microenvironment. A. Representative images (200X) of immunoflourescence staining of B16F1 tumor infiltrating CD4 and CD8 T cells as well as $CD11b^+$ myeloid cells. B. The number of T and myeloid cells in each field of view was counted and averaged from 6 fields of 2 independent experiments. * denotes a significant difference (p < 0.05) between WT and *p53^{null}* mice. Two-tailed Student's t-test.

Supplemental Figure S2. *Trp53* inactivation in B16F1 tumor bearing hosts does not impose significant effects on the splenic effector T cell function as compared with those in WT tumor bearing mice. Splenocytes were harvested from B16F1 tumor bearing WT and $p53^{null}$ mice. A. The CD4 effector function as their capacity for producing effector cytokines was examined via intracellular staining. B. The frequency of FoxP3⁺ regulatory T cells was also determined via FACS. Representative FACS plots of 5-6 mice from two independent experiments.

Supplemental Figure S3. Tumor associated MDSCs from WT and $p53^{null}$ mice share similar phenotype and suppressor function on per cell basis. TIL-MDSCs were harvested from enzymatically digested tumors and enriched via CD11b positive selection via magnetic beads. (A) The heterogeneity of MDSCs was confirmed via histology examination of cytospinned cells. (B) The suppressor function of MDSC was determined as their capacity in suppressing the proliferation of α -CD3 and α -CD28 stimulated T cells at various T cell to MDSC ratios. Results are presented as mean \pm standard error (s.e.) of 4 - 5 mice from at least two independent experiments.

Supplemental Figure S4. Analyses of cell surface marker expression profile of MEF from $p53^{null}$ mice. Fibroblasts were harvested from $p53^{null}$ embryos. Their surface marker expression was examined via FACS. Isotype controls are shown as open histograms.

Supplemental Figure S5. Differential differentiation of BM-MDSCs from WT and $p53^{null}$ mice sstimulated by different myeloipoietic cytokines. A. BM cells from WT and $p53^{null}$ mice were differentiated to MDSCs in a 4-day culture in the presence of 100 ng/ml G-CSF+10 ng/ml IL-6 or 100 ng/ml G-CSF+ 250 Ug/ml GM-CSF. The composition of M-MDSC and G-MDSCs was examined via FACS. The efficacy of MDSC differentiation from WT and $p53^{null}$ BM cells under each condition was examined and compared. B. The cellular phenotype of BM-MDSCs differentiated under different cytokine conditions was examined.

Supplemental Figure S6. SPSCs from $p53^{null}$ mice enhanced BM-MDSCs differentiation in part by suppressing apoptosis of myeloid precursors. BM cells from WT and $p53^{null}$ mice were differentiated to MDSCs in a 4-day culture with 100 ng/ml G-CSF+10 ng/ml IL-6 in the absence or presence of SPSCs from $p53^{null}$ mice. The percentage of apoptotic (7-AAD⁺) cells was determined at day 2 of the culture (n=3). * denotes a significant difference (p < 0.05) between groups with and without SPSCs. Two-tailed Student's t-test.

Guo, G. et al.







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