**SUPPLEMENTARY FIGURE LEGENDS**

***Figure S1:*** **Characterization of the TCR transgenic h3T *vs.* h3T-*p53* KO mouse**. ***A)*** Generation of h3T-*p53* KO was done by crossing *p53-*KO with h3T transgenic mouse, followed by back crossing the F1 heterozygotes to the *p53*-KO parent. Lane 1-3 shows the genotyping of an h3T mouse evaluating the expression of TCR-α chain, TCR-β chain, and *p53*+/+ (450 bp) respectively. Lane 4-6 shows the genotyping data from an h3T-*p53*+/- with TCR-α chain, TCR-β chain, and *p53*+/- (450 bp, and 650 bp) respectively. Lane 7-9 shows a representative genotyping from h3T-*p53* KO mouse with TCR-α chain, TCR-β chain, and p53-/- (650 bp) only. ***B)*** Splenocytes from h3T and h3T-*p53* KO transgenic mice were stimulated overnight using the surrogate antigen presenting T2-A2 cells loaded with cognate human tyrosinase peptide antigen (1 μg/ml) or control human Mart-1 peptide antigen (1 μg/ml) before co-staining with fluorochrome conjugated anti-CD25 and anti-CD69 antibody. The data was analysed using FACS. ***C)*** The spleens obtained from the h3T and h3T-*p53* KO mouse at eight weeks of age was pictured. ***D)*** TCR activated splenic T cells from h3T and h3T-*p53* KO mouse were re-stimulated with cognate human tyrosinase antigen pulsed T2-A2 cells (*upper panel*) or control peptide Mart-1 pulsed T2-A2 cells (*lower panel)* for 4 hr, before staining was carried out using fluorochrome conjugated Annexin V and subsequently analysis was done using FACS. The red box compares the Annexin Vlo cells (< 102 MFI). Numerical value represents percent positive gated cells. All experiments were repeated at least thrice with similar results. ***E)***. Splenic T cells from wild type (WT) and *p53*-KO mouse that were TCR activated using anti-CD3/anti-CD28 antibody in presence of IL2 (50 IU/ml) for three days were used to obtain RNA for quantitative real time PCR analysis and expression of key glycolytic pathway genes.

***Figure S2:*** **Reduced *p53* levels in T cells enhance tumor control while inhibiting exhaustion phenotype and iTreg generation.** ***A)*** Representative pictures of the C57BL/6 recipient mice that were treated by adoptively transferring either 106 h3T or h3T-*p53* KO T cells to control B16-F10 tumor growth. ***B)*** TCR activated T cells (with 100U/ml IL2) in presence or absence of pifithrin-α (5μM; present though out the culture) plus pifithrin-μ (5μM; present only for last 4h) were used for the evaluation of: (*i*) intracellular reactive oxygen species (using DCFDA) and nitric oxide (using DAF), (*ii*) CD95 and CD95L expression, and (*iii*) expression of exhaustion markers Lag-3, PD1 using flow-cytometry. Numerical values indicate the mean fluorescence intensity for each histogram overlays, and blue histogram in the overlay is for WT and red for *p53* inhibitor treated T cells. ***C)*** Human peripheral blood T cells from normal healthy individuals were either left untreated or were pretreated with *p53* inhibitors pifithrin-α (30μM) or pifithrin-μ (10μM) before culturing them under *ex vivo* iTreg programming conditions for three days. Cells were thereafter washed and stained for FoxP3 expression and data was acquired using FACS. *N*=3.