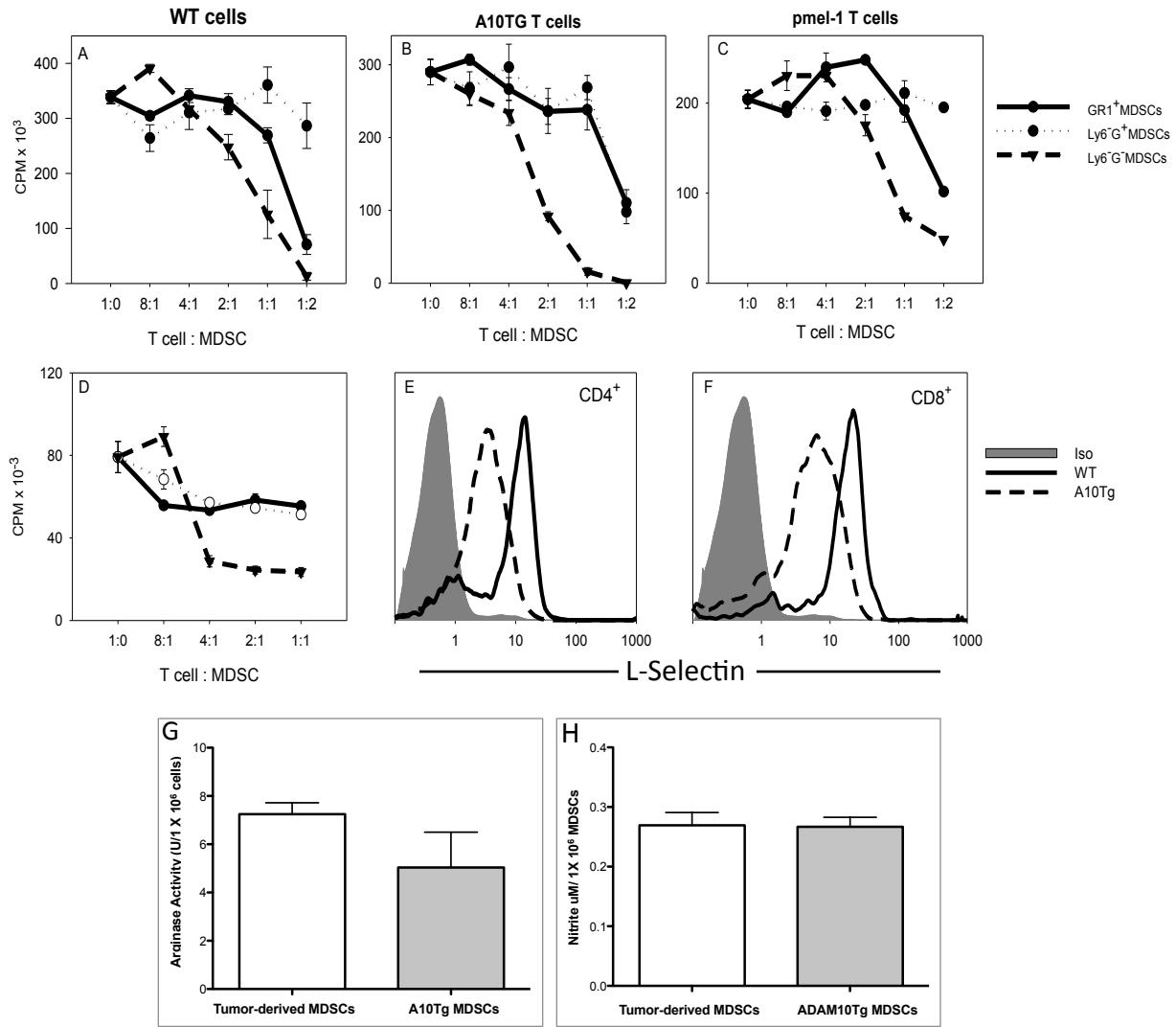
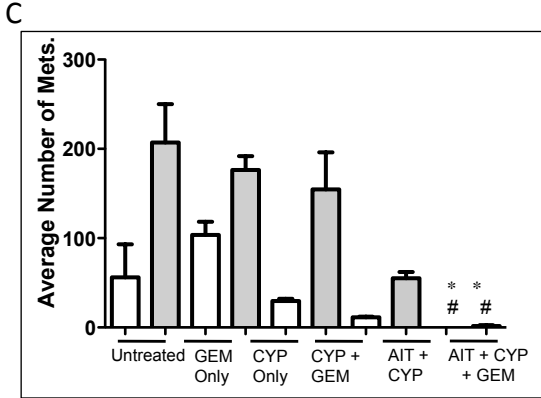
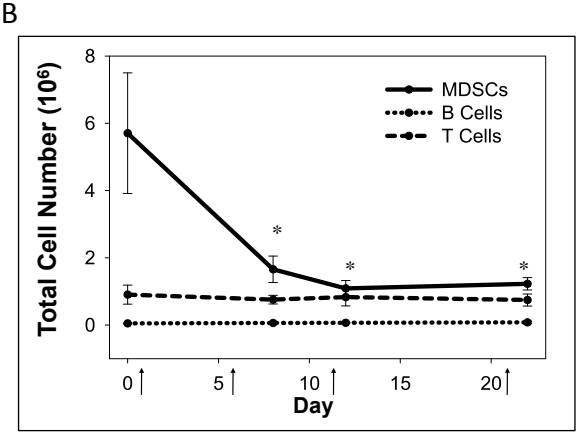
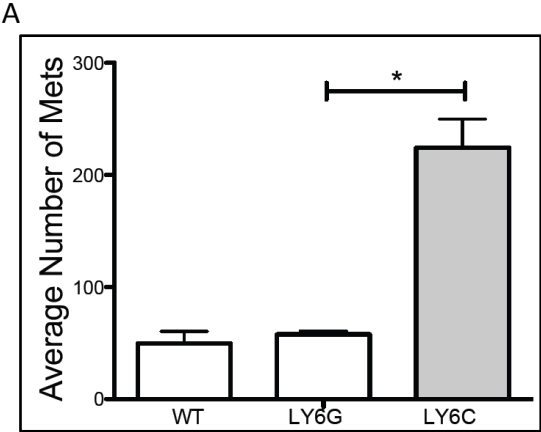


Supplementary Figure 1



Supplementary Figure 1. MDSCs from ADAM10Tg mice are phenotypically and functionally analogous to tumor-derived MDSCs. (A) Proliferation of WT and ADAM10Tg (B) T cells or (C) pmel-1 TCR transgenic splenocytes in the presence of increasing amounts of CD11b⁺ MDSCs (Ly6G⁺, Ly6G⁻, or Gr-1⁺); WT and ADAM10Tg T cells are stimulated with immobilized anti-CD3 and soluble anti-CD28. (D) Tumor derived MDSCs were purified from LLC bearing mice and used in suppression assays with Pmel1 splenocytes at increasing ratios (T cells or splenocytes: MDSCs, *p<0.05). All Pmel-1 splenocytes were stimulated with soluble gp100. (E) Cell surface expression of L-selectin (CD62L) by CD4⁺ and CD8⁺ (F) gated T cells from peripheral lymph nodes (PLNs). Lysates of MDSCs derived from Lewis Lung Carcinoma bearing WT hosts and ADAM10Tg MDSCs were analyzed for the activity of (G) Arginase by urea production and (H) Nitric oxide by Greiss Reagent. The data is representative of at least three independent experiments with splenocytes from three or more mice.

Supplementary Figure 2



Supplementary Figure 2. Gemcitabine selectively depletes MDSCs, which allows for effective AIT with tumor specific T cells. (A) Quantification of B16 lung metastasis in WT C57 with AT of either granulocytic (CD11b⁺Ly6G⁺) or monocytic (CD11b⁺Ly6C⁺) MDSCs. (B) Cytometric analysis of peripheral blood leukocyte levels in ADAM10Tg mice following *i.p.* injections with gemcitabine (upward arrow) every five days for three weeks, *p<0.05. (C) Number of B16 lung metastases in mice treated with AIT comprised of pmel-1 transgenic T cells and chemotherapeutics as described in *Methods*. More than five mice were used per group in three independent experiments. *p<0.05 in comparison to respective untreated controls and #p<0.05 in comparison to respective AIT+ CYP treatment.