

Fig. S1. Loss of STAT2 and STAT1 protein expression occurs in the tumor. Splens and tumors harvested at day 7 from tumor-bearing mice were labeled for STAT1 (green) and STAT2 (red). Nucleus was stained with DAPI (blue). Shown are representative images of tumors (inside and periphery sections). Images of B16-F1 tumor cells grown *in vitro* and spleen sections are shown as a controls.

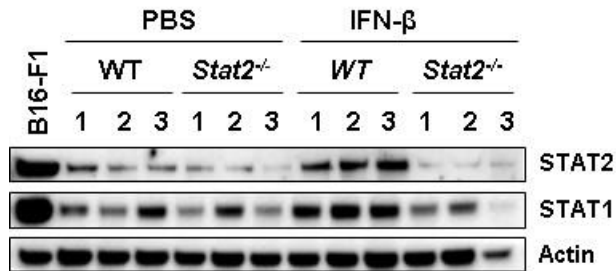


Fig. S2. STAT1 and STAT2 expression within tumors is reestablished only in WT mice treated with IFN- β . Western blot analysis of three individual tumors shows rescued expression of STAT1 and STAT2 only in tumors established in WT but not in *Stat2*^{-/-} mice with IFN- β intervention. Actin served as an internal loading control.

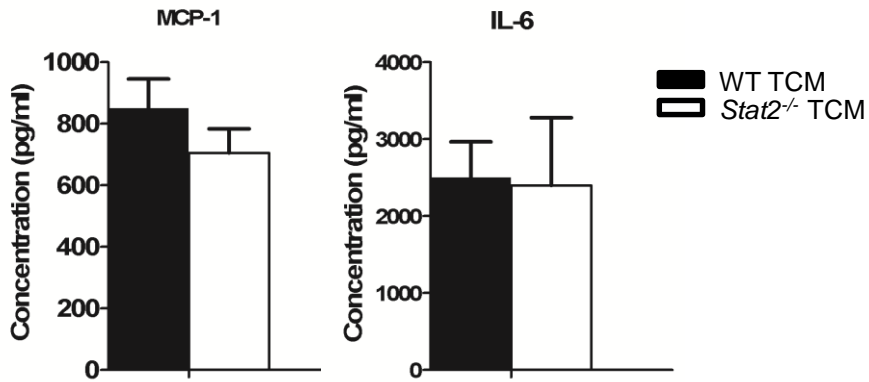


Fig. S3. Production of MCP-1 and IL-6 by tumors established in WT or *Stat2*^{-/-} mice is similar. Tumor conditioned medium (TCM) prepared from individual tumors (n=6) were analyzed using BD™ Cytometric Bead Array (CBA) Kit for six different inflammatory cytokines. MCP-1 and IL-6 levels are shown. IFN- γ and TNF- α were similar but detected at low levels. IL-10 and IL-12 were undetected.

Table S1. Genes downregulated in B16-F1 tumors established in *Stat2*^{-/-} mice compared against WT control mice

Accession #	Gene Name	Gene Symbol	Fold change
DOWNREGULATED:			
NM_008329	interferon activated gene 204	IFI204	3.37
NM_008599	(C-X-C motif) ligand 9	CXCL9	3.23
ENSMUST00000165774	guanylate binding protein 2, interferon inducible	GBP2	2.32
NM_009465	AXL receptor tyrosine kinase	AXL	2.31
NM_013532	leukocyte immunoglobulin-like receptor, subfamily B, member 4	LILRB4	2.27
NM_001162910	predicted gene 3604	GM3604	2.04
NM_010240	ferritin light chain 1	FTL1	2.02
NM_008326	immunity-related GTPase family M member 1	IRGM1	1.98
NM_010634	fatty acid binding protein 5, epidermal	FABP5	1.95
NM_001170853	myeloid nuclear differentiation antigen like	MNDAL	1.93
NM_009777	complement component 1, q subcomponent, beta polypeptide	C1QB	1.93
NM_001267695	cathepsin S	CTSS	1.89
NM_020557	cytidine monophosphate (UMP-CMP) kinase 2, mitochondrial	CMPK2	1.88
ENSMUST00000147599	Retinol dehydrogenase 13	RDH13	1.87
NM_001042605	CD74 antigen	CD74	1.86
NM_001113326	macrophage scavenger receptor 1	MSR1	1.84
NM_021443	chemokine (C-C motif) ligand 8	CCL8	1.83

Relative transcript levels are presented as fold change and are the average of three individual tumors with a false discovery rate cut off of 0.05 and $p < 0.001$. Thirty one genes were found downregulated in tumors that formed in *Stat2*^{-/-} mice. Seventeen genes showed a fold change decrease of >1.8 ; of which 12 participate in immune responses. Twenty-two genes were found upregulated. Eight genes showed a fold change increase of >1.8 . These genes encode for olfactory receptors. Student's *t*-test was applied for comparison between the two groups to assess significance. The complete microarray data set can be found at NCBI GEO, accession number GSE56792

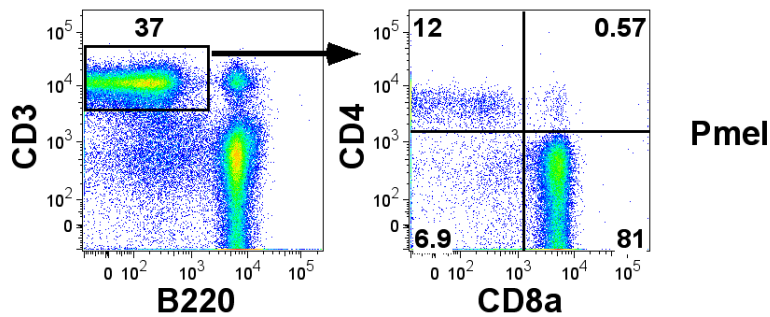
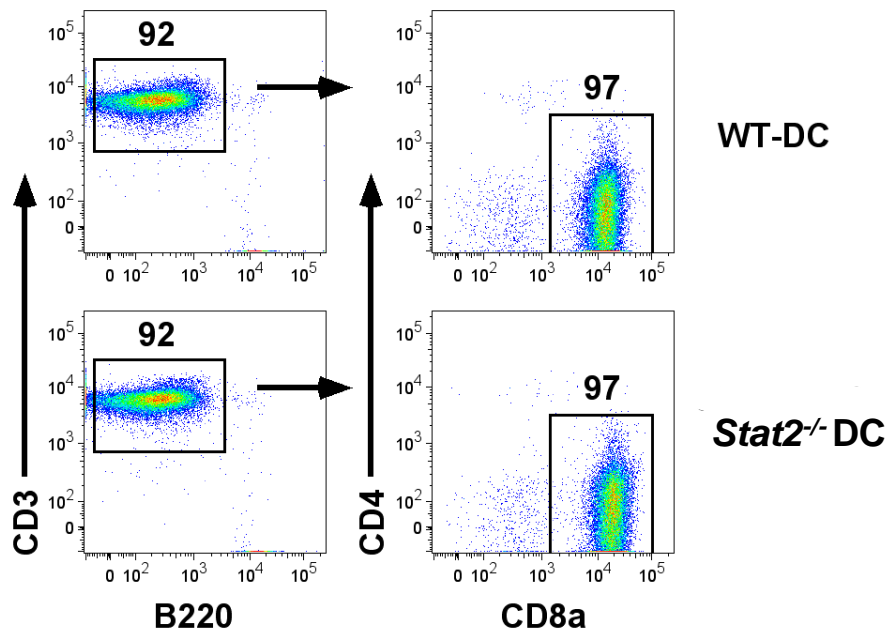
A**B**

Fig. S4. Pmel-1 CD8⁺ T lymphocytes activated *in vitro* by WT or *Stat2*^{-/-} BMDCs. Pmel-1 lymphocytes were stained before (A) and five days after (B) *in vitro* activation with WT or *Stat2*^{-/-} BMDCs stimulated with LPS and pulsed with human gp100 peptide. Surface markers CD3, B220, CD4, and CD8 were detected by flow cytometry. Dot plots show frequencies of gated populations in Pmel-1 lymphocytes. Data are representative of two experiments.

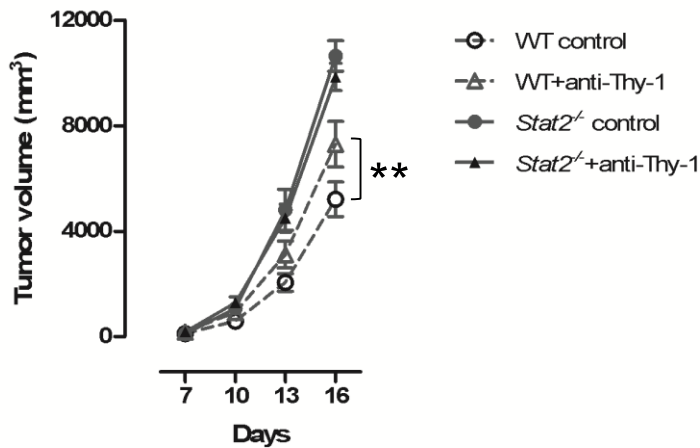
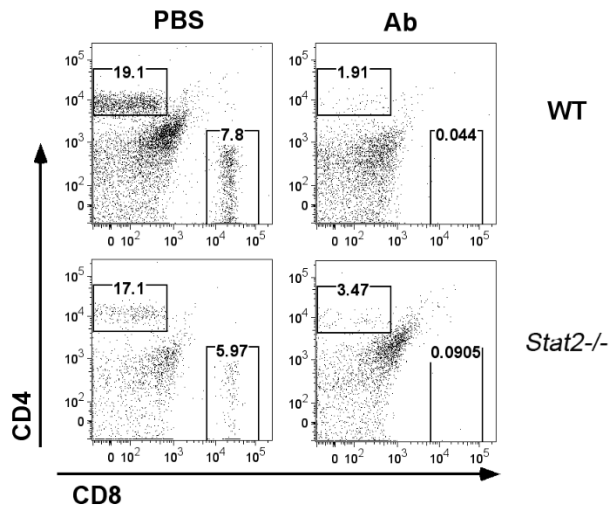
A**B**

Fig. S5. Contribution of non-T cell immune effectors that require STAT2 for the development of antitumor immunity. (A) WT and *Stat2*^{-/-} mice were depleted of T cells with anti-pan T-cell antibody (α -Thy-1) prior to tumor challenge and during the course of the study. T cell depletion in WT mice promoted an increase in tumor size but not to the same degree as observed in *Stat2*^{-/-} mice. (B) Dot plots confirm T-cell depletion with anti-Thy-1 (Ab) in WT and *Stat2*^{-/-} mice compared to control group (PBS).