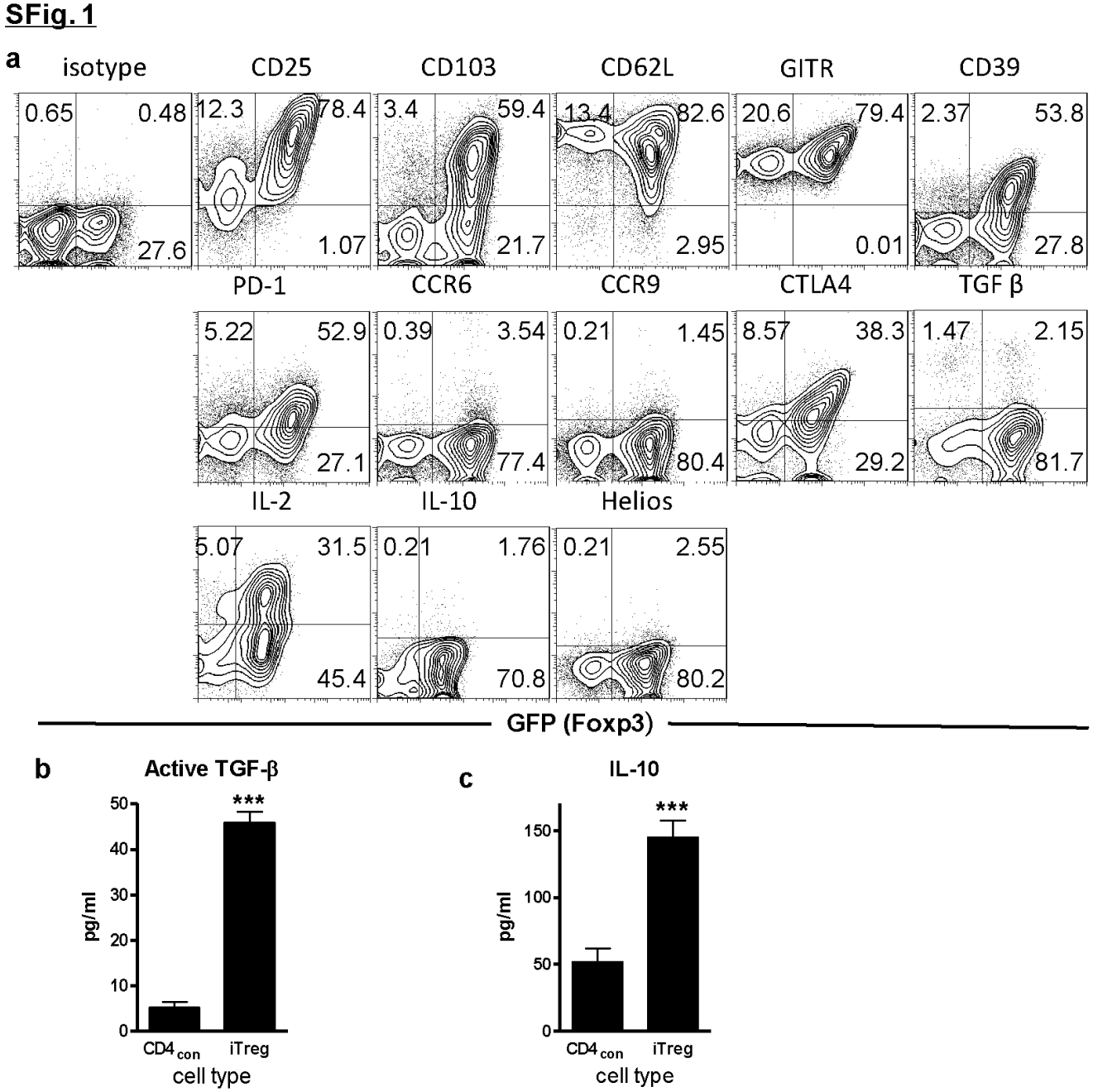
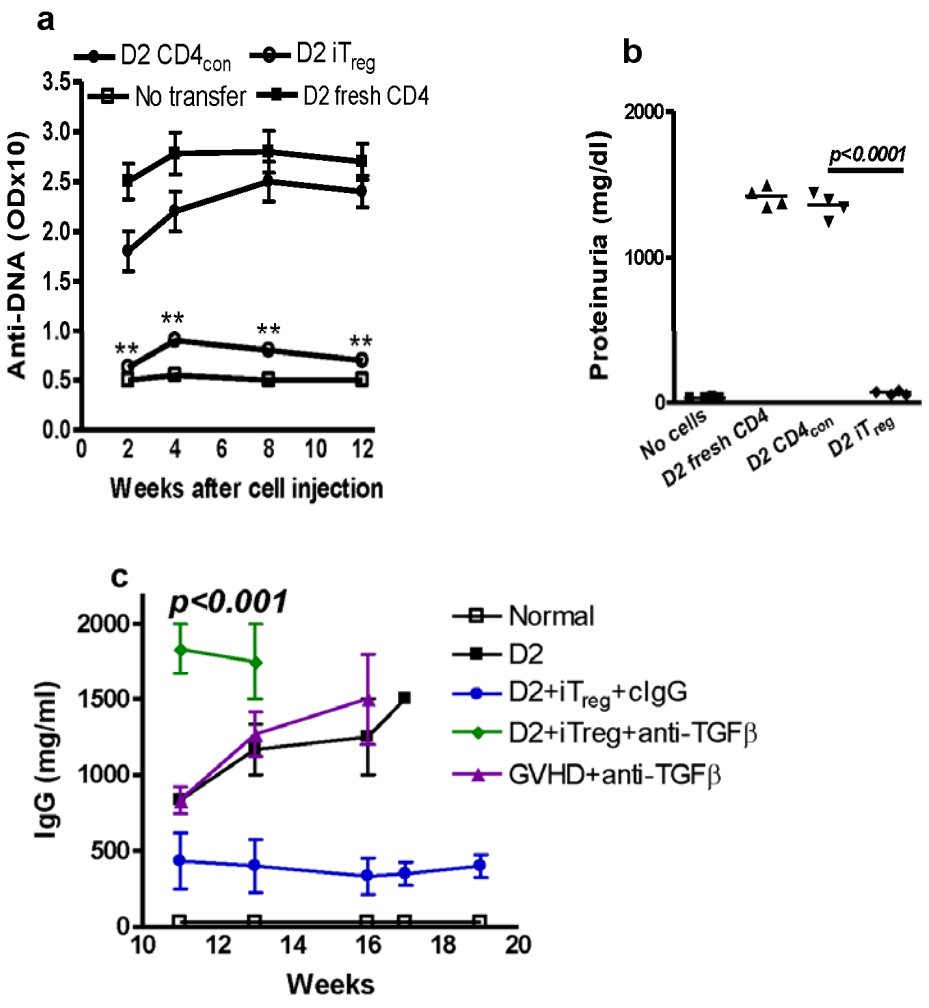
Supplementary Figure S1 iTreg cell phenotype and cytokine profile. (A) iTreg were induced as described in the Methods section and the expression of Treg-related molecules was analyzed by flow cytometry. Data is representative of four independent experiments. (B-C) $CD4_{con}$ or iTreg generated as above were re-stimulated with anti-CD3/CD28 coated beads (1:5 ratio) and IL-2 (20 U/ml) for three days and supernatants were subjects to ELISA. Values are mean ± SEM of triplicate samples and data is representative of three separate experiments.

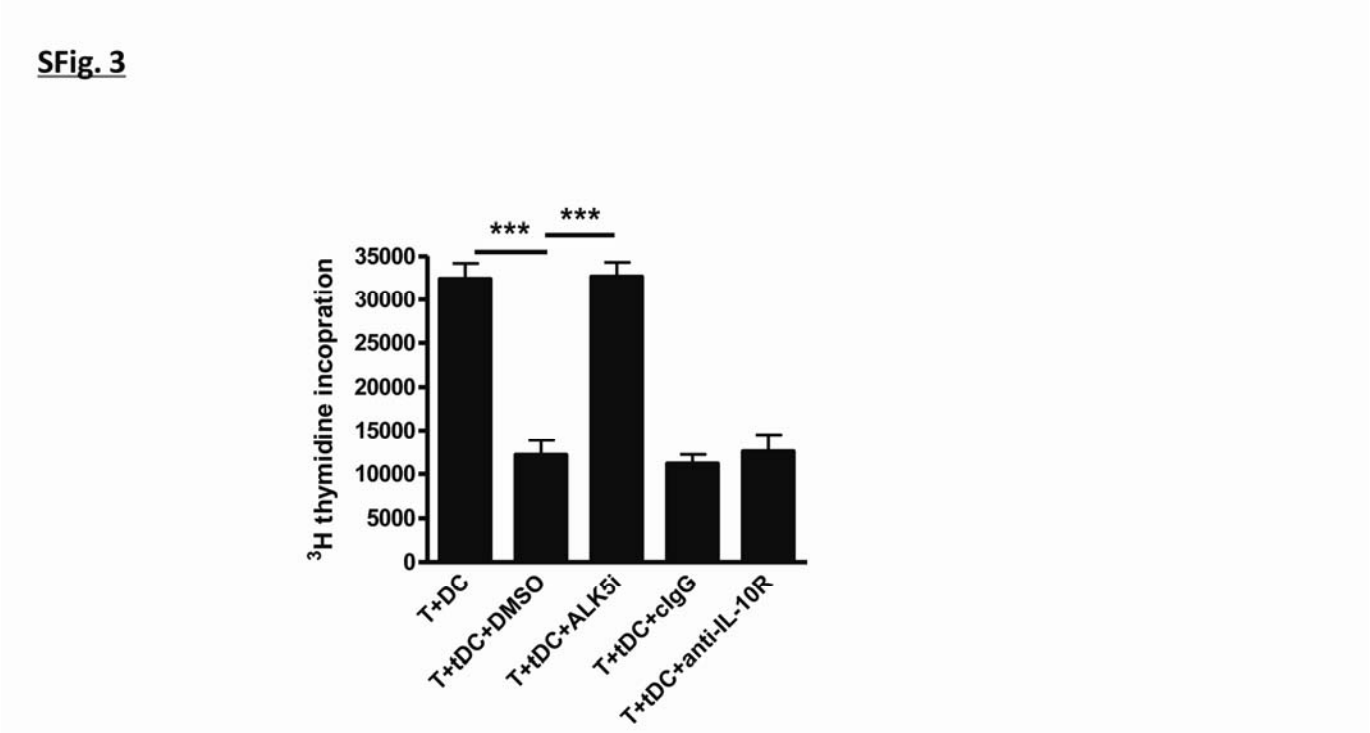


Supplementary Figure S2 iTreg induced from D2 cells are not pathogenic and suppress cGVHD via TGFβ-dependent mechanism. 12x10^6 fresh, $CD4_{con}$ or iTreg induced from D2 $CD4^+$ cells were transferred into D2B6F1 mice. (A) The levels of anti-dsDNA antibodieswere evaluated weekly as indicated. (B) The proteinuria was assessed 12 weeks post-transfer (n=5 in each cohort). $^{**}p<0.01$, iTreg vs. $CD4_{con}$ or fresh $CD4^+$ cells. (C) 12x10^6 fresh $CD4^+$ D2 cells were transferred alone or with 5x10^6 i*Treg* into D2B6F1 mice. In some groups, anti-TGFβ antibody or control IgG were administered. IgG levels were examined by ELISA. Five mice in each cohort were included and data were combined from two independent experiments. $P<0.001$, anti-TGFβ vs control IgG.

Sfig. 2

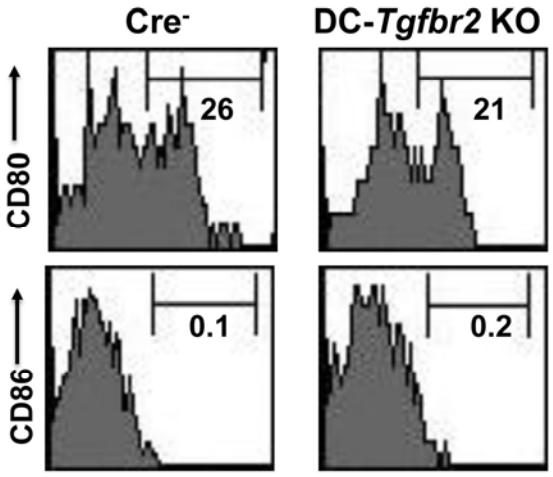


Supplementary Figure S3 DCs primed with iTreg decrease antigen-presenting capacity via TGFβ but not IL-10 signaling pathway. Splenic $CD11c^+$ cells in C57BL/6 mice were co-cultured with $CD4_{con}$ or iTreg cells in the presence of ALK5i or DMSO, or anti-IL-10R or control IgG for three days as above. $CD11c^+$ cells were sorted and added $CD25^+$-depleted T cells from D2 mice and T cell proliferation was measured by ^{3}[H]-incorporation. Values are mean ± SEM of triplicate samples and data is representative of four separate experiments.

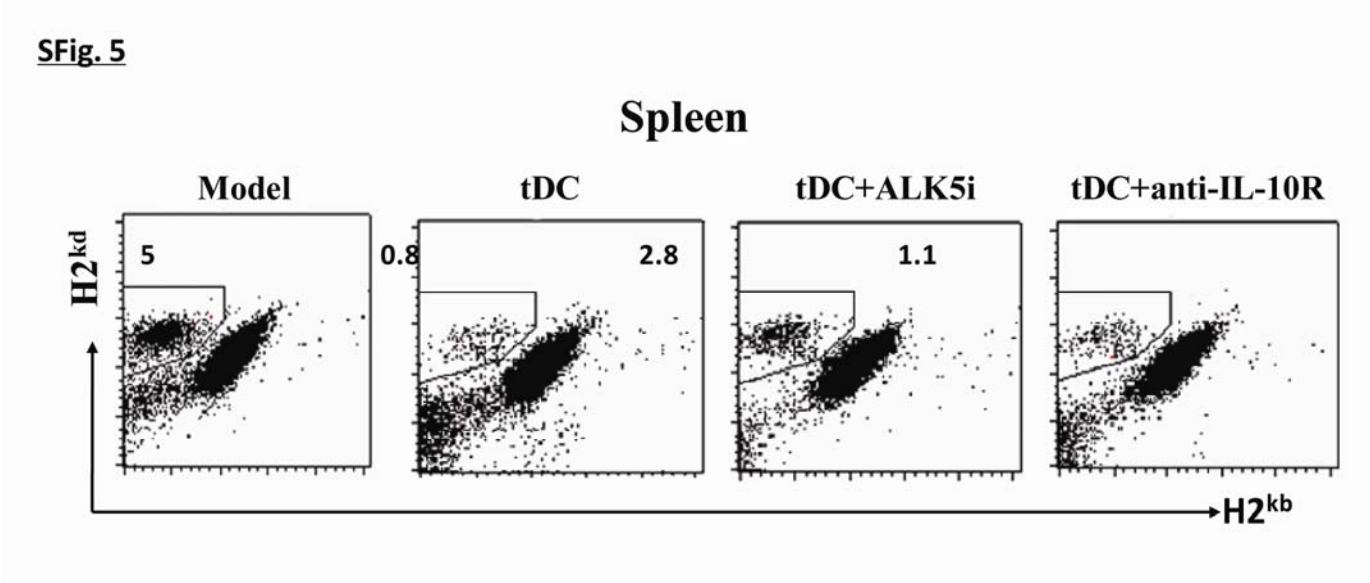


Supplementary Figure S4 Comparable CD80 and CD86 expression on $CD11c^+$ DC cells between Cre^- (wild type) and Cre^+ (DC-*Tgfbr2* KO) littermates. CD80 and CD86 expression in splenic $CD11c^+$ cells from naïve Cre^- and DC-*Tgfbr2* KO mice. Data are representative of five mice in each strain.

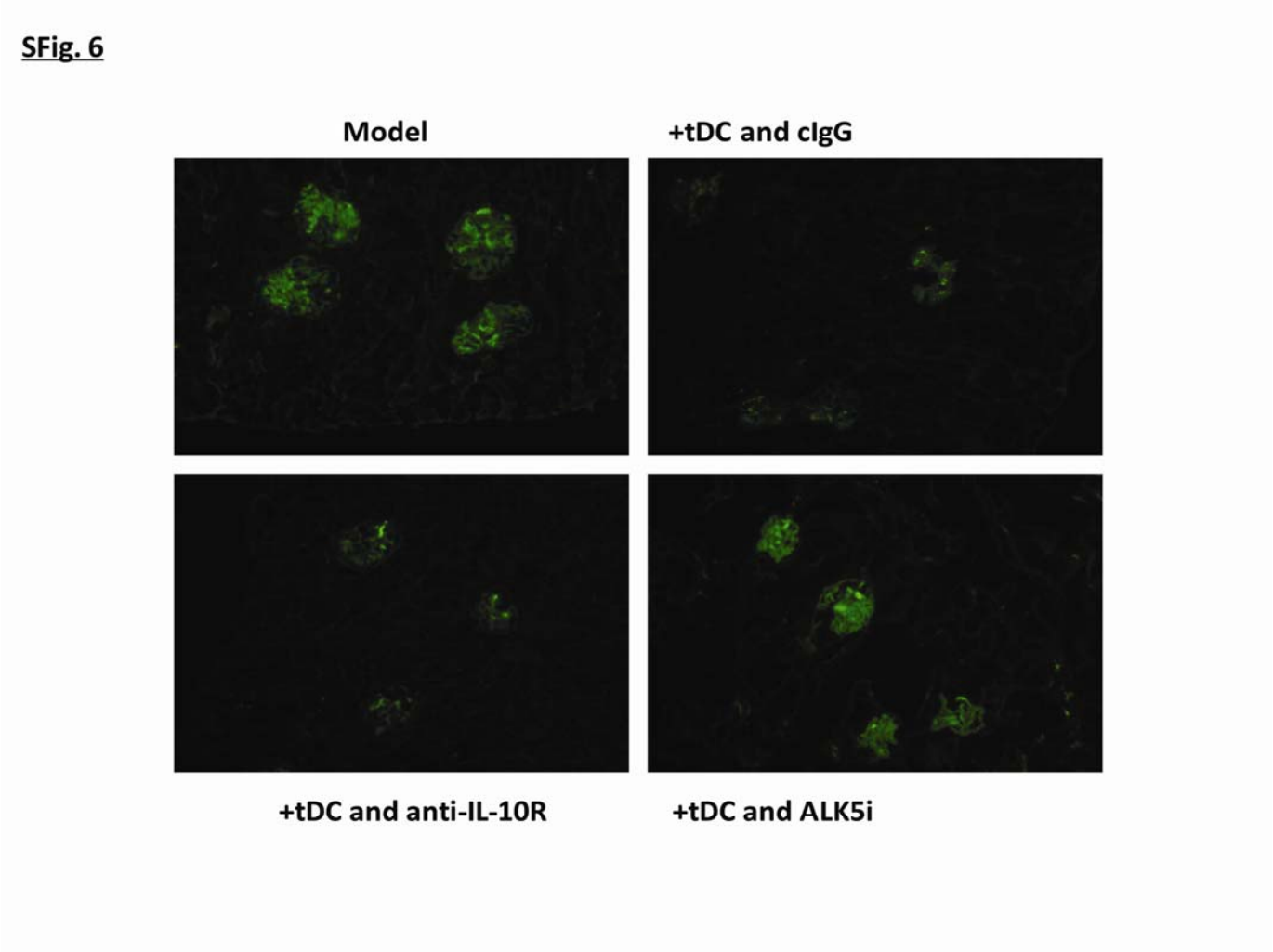
SFig. 4



Supplementary Figure S5 Tolerogenic DCs suppress donor engraftment in cGVHD mice. $5x10^5$ $CD11c^+$ cells were sorted from lupus mice treated with iTreg (tDCs) and were co-transferred with $12x10^6$ fresh D2 $CD4^+$ cells into D2B6F1 mice.ALK5i or DMSO (vehicle), anti-IL-10R or control IgG were administered in separate groups. The size of donor engraftment ($H2^{kd+}$ and $H2^{kb-}$) was analyzed by flow cytometry 3 weeks post-transfer. Data is representative of two independent experiments with total of 10 mice in each cohort.



Supplementary Figure S6 Tolerogenic DCs suppress renal IgG deposition in cGVHD mice. $5x10^5$ $CD11c^+$ cells were sorted from lupus mice treated with i*Treg* (tDCs) and were co-transferred with $12x10^6$ fresh D2 $CD4^+$ cells into D2B6F1 mice. ALK5i or DMSO (vehicle), anti-IL-10R or control IgG was administered in separate groups. The IgG deposition in kidney of cGVHD mice 20 weeks post-transfer was examined by fluorescence microscopy. Data is representative of two independent experiments with total of 10 mice in each cohort.



Supplementary Figure S7 Tolerogenic DCs suppress lupus through TGFβ- but not IL-10-dependent mechanism. iTreg were co-cultured with DCs from Cre^- or DC-*Tgfbr2* KO mice for three days. These DCs ($2x10^5$) were then co-transferred with naïve $CD4^+CD45RB^{high}$ cells ($5x10^5$) into $Rag1^{-/-}$ mice. Mice were sacrificed 4 weeks post-transfer. MLN cells were analyzed for TNFα and IFNγ mRNA expression using real-time PCR. All experiments were repeated at least twice with similar results.