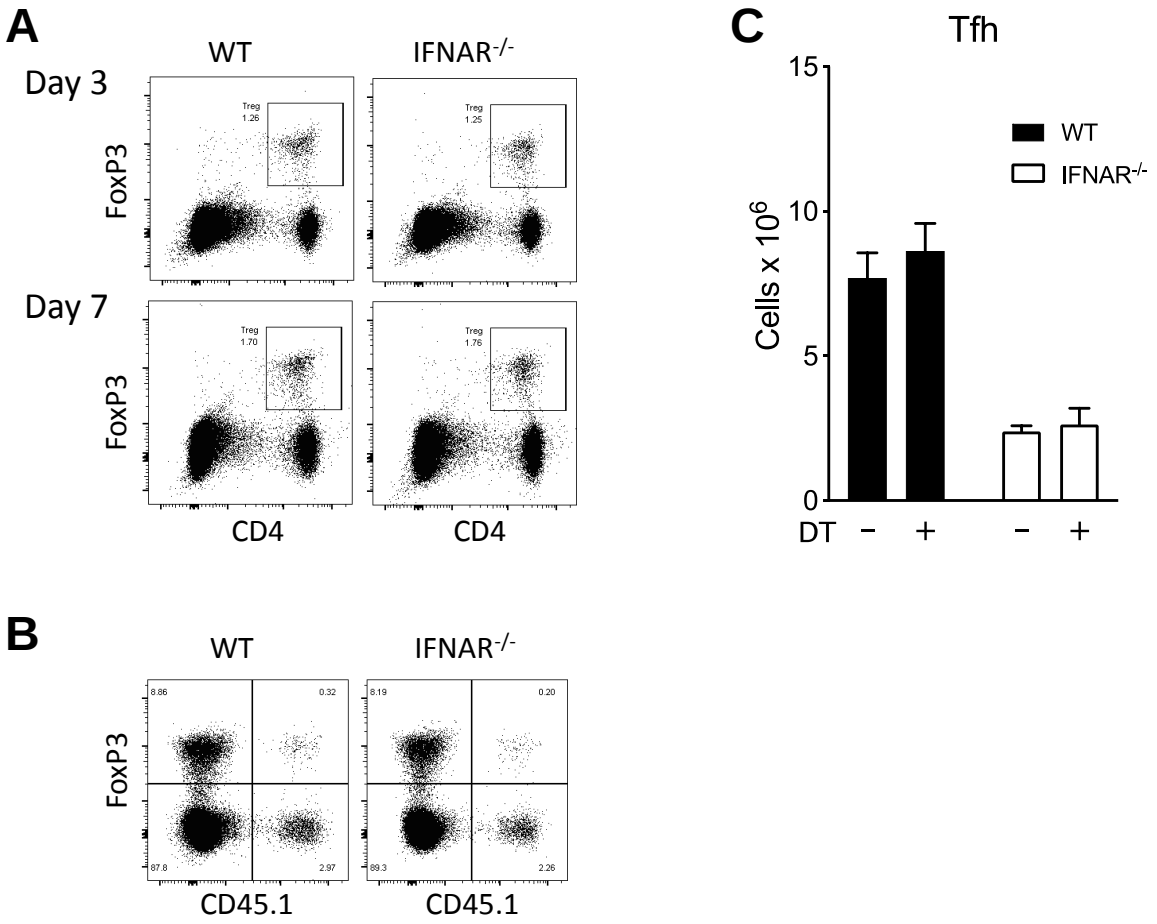
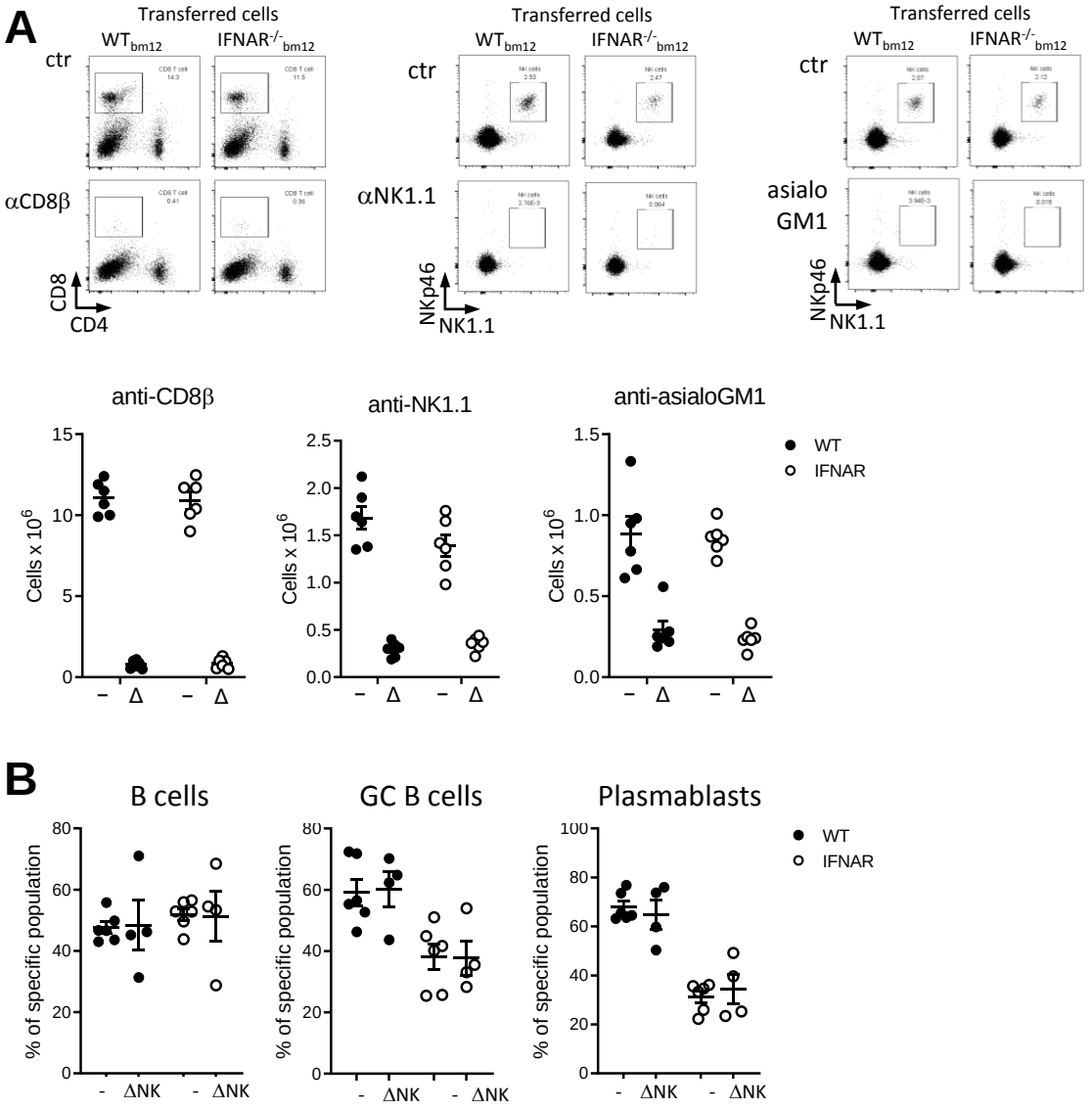
Supplemental Figure 1; Klarquist et al.



Supplemental Figure 1. No role for Tregs in the attrition of IFNAR-/- TFH.

A. WT or IFNAR-/- CD45.1 CD4bm12 T cells CD4bm12 T cells were transferred into CD45.2 intact recipients. The Treg population (live CD4+FoxP3+CD25+) was assessed in total spleen 3 and 7 days after transfer. Plots are gated on live total splenocytes. **B**. Treg assessment in recipient and transferred CD4 T cell population 5 days after transfer of WT or IFNAR-/- CD45.1 CD4bm12 T cells. Dot plots show Foxp3 vs CD45.1 in the live CD4+ population. **C**. WT (black bar) or IFNAR-/- (white bar) CD45.1 CD4bm12 T cells were transferred into Foxp3-DTR mice. Mice were treated with vehicle (-) or DT (+) one day before and 3 and 10 days after T cell transfer. Absolute numbers of donor TFH in each graft was assessed and shown as mean ± SEM with n=4/group, where *p≤0.05.

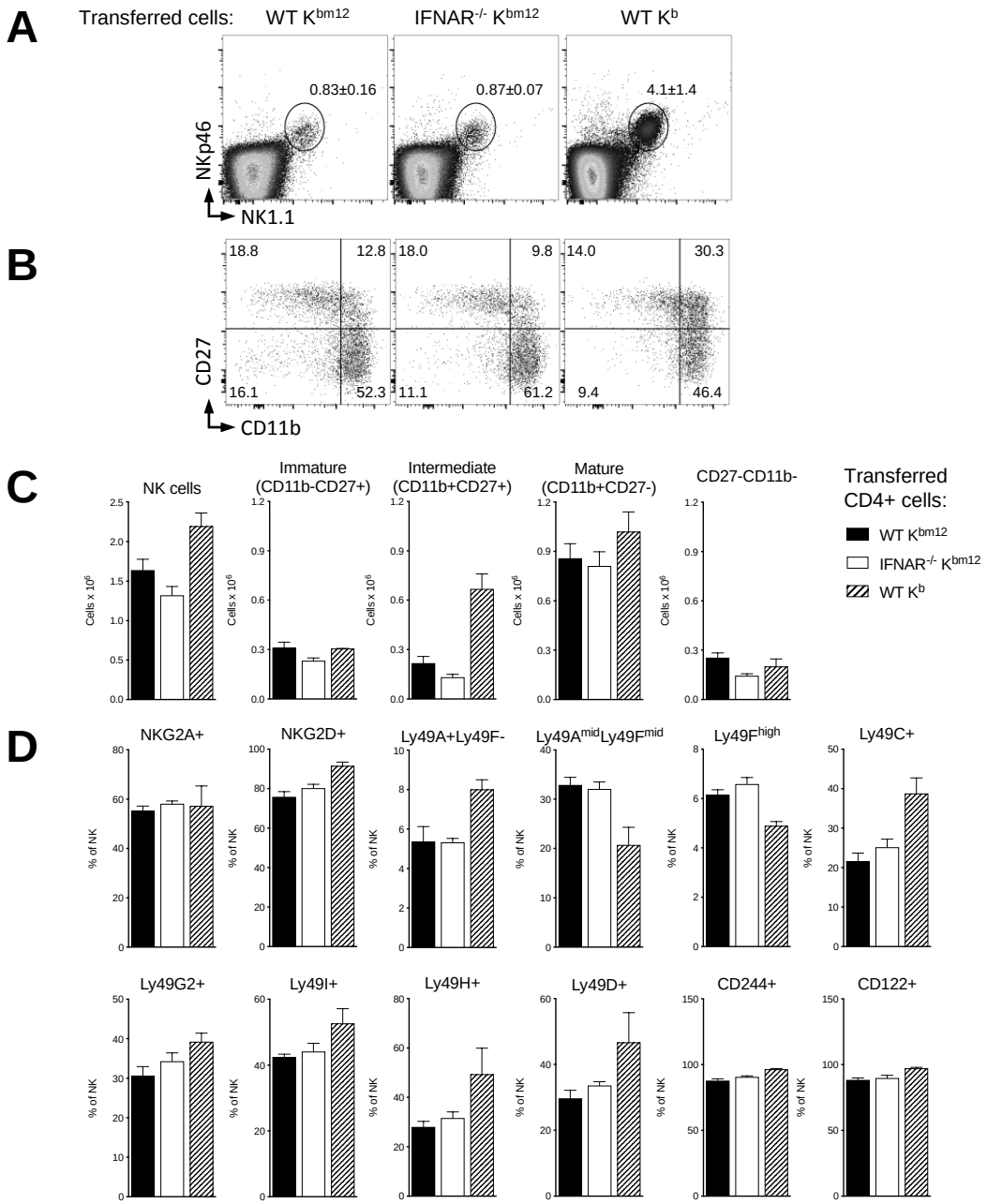
Supplemental Figure 2; Klarquist et al.



Supplemental Figure 2. NK cell depletion does not alter the dependency of B cells on type I IFN.

A. Representative example of depletion efficacy of depletion of the different cell populations in mice receiving either WT or IFNAR-/- CD4bm12 T cells. **B**. WT (CD45.2, black bar) and IFNAR -/- (CD45.1, white bar) bone marrow was mixed in a 1:1 ratio and transplanted into irradiated WT recipients. After 12 weeks, mice were left untreated or NK-depleted with anti-asialo-GM1 before transfer of WT CD45.1/2 CD4bm12 T cells. B cell responses were assessed 2 weeks later. WT and IFNAR-/- B cells grafted in a 1:1 ratio. As seen in figure 2, IFNAR-/- B cells showed significantly reduced numbers of GC B cells and plasmablasts and this reduction could not be overcome by depletion of NK cells. Data are shown as mean ± SEM with n=5-6/group, where *p≤0.05.

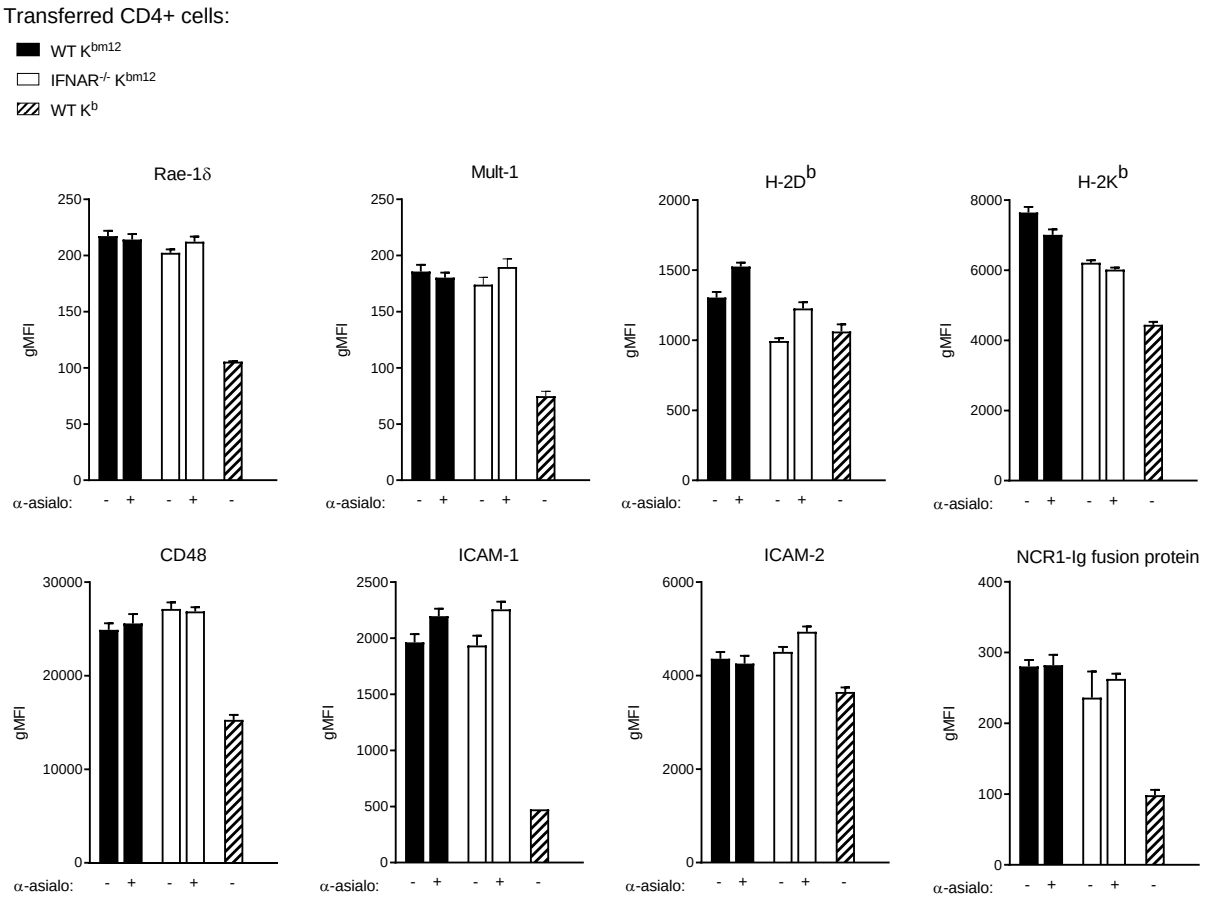
Supplemental Figure 3; Klarquist et al.



Supplemental Figure 3. Numbers and phenotype of splenic NK cells in the bm12 model of SLE.

Congenically marked (CD45.1+) WT $CD4_{Kbm12}$ (black circles/bar), IFNAR-/- (white circles/bar) $CD4_{Kbm12}$, or WT $CD4_{Kb}$ T cells (hatched bars) were transferred into CD45.2 WT mice. After 2 weeks the number of NK cells and expression of NK activating and inhibitory receptors in each recipient was assessed. **A.** Representative plots for NK cell determination, gated on live CD3− lymphocytes, where numbers indicate the average percentage of live splenocytes positive for NK1.1 and NKp46 for each group ± SEM. **B.** Representative CD27 x CD11b plots for NK1.1+NKp46+ NK cells, where numbers indicate the averages for each group. **C.** Total splenic NK cell numbers. **D.** Expression of inhibitory and activating receptors as a % of NK cells. Data are expressed as mean ± SEM with n=2-5/group.

Supplemental Figure 4; Klarquist et al.



Supplemental Figure 4. WT and IFNAR$^{-/-}$ TFH cells express similar levels of several known NK activating and inhibitory proteins.

Congenically marked (CD45.1+) WT CD4$_{Kbm12}$ (black circles/bar), IFNAR-/- (white circles/bar) CD4$_{Kbm12}$, or WT CD4$_{Kb}$ T cells (asterisks/hatched bars) were transferred into intact or NK-depleted (anti-Asialo-GM1) CD45.2 WT mice. Five days later, geometric mean fluorescent intensity (gMFI) was analyzed for various NK cell activating and inhibitory molecules on donor TFH. Data are expressed as mean ± SEM with n=2-5/group.