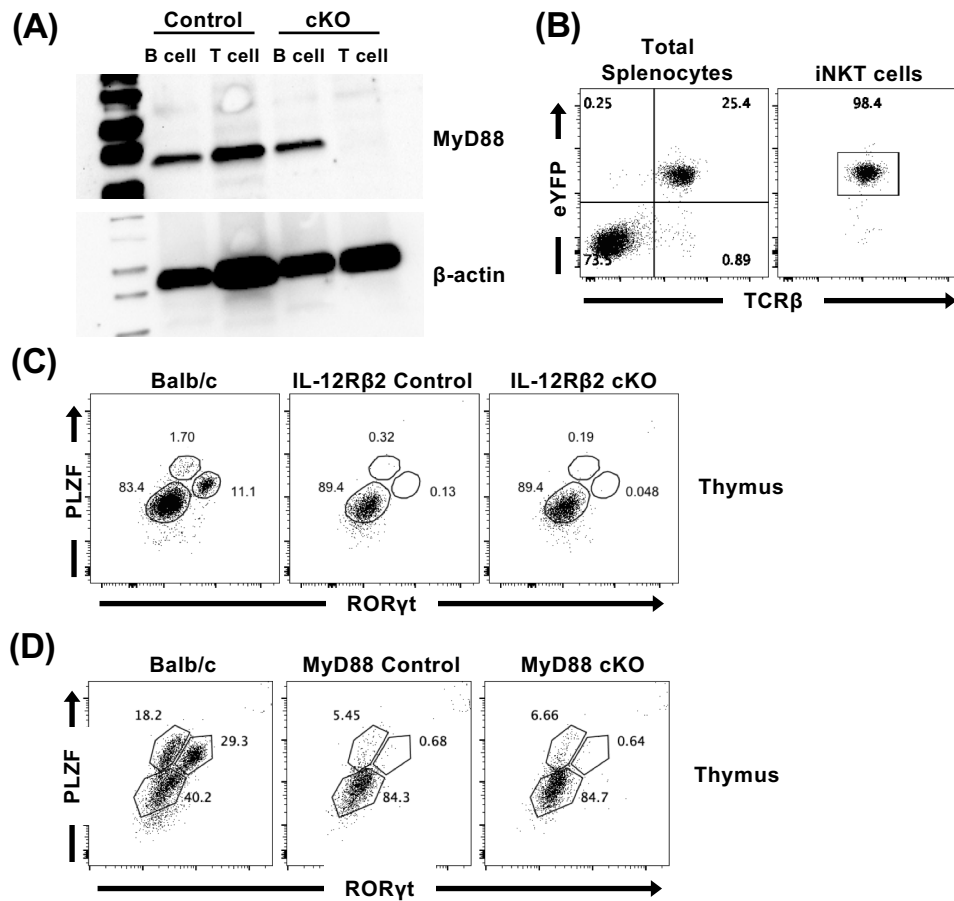
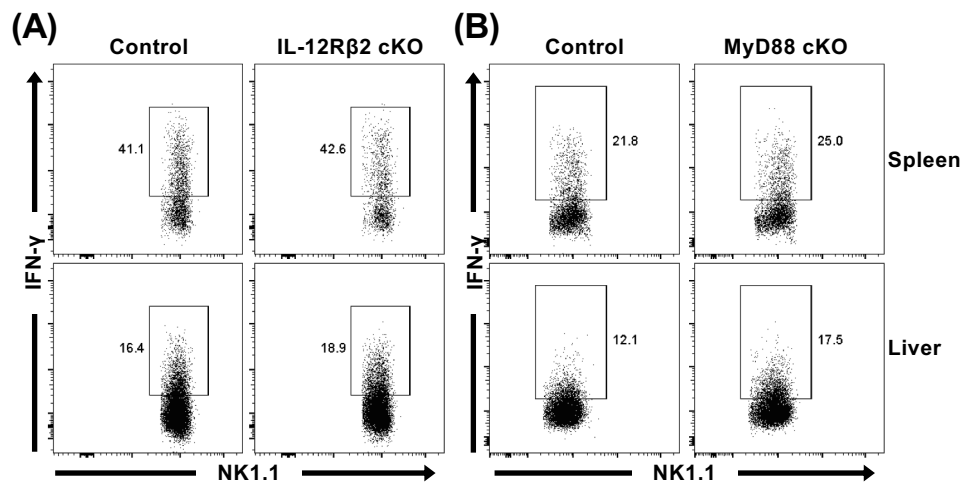


Supplementary Figure 1



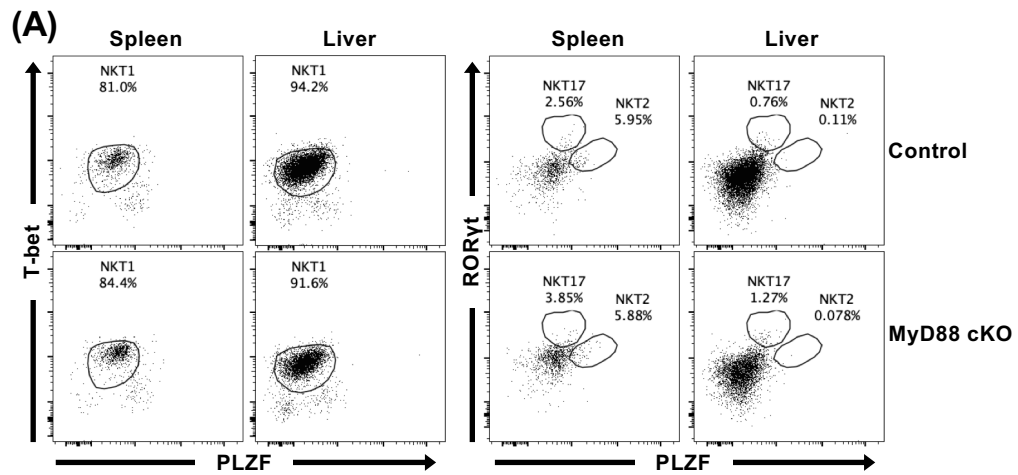
Supplementary Figure 1. IL-12R β 2 and MyD88 signaling are dispensable for naïve NKT cell lineages. (A) MyD88 and β -actin protein expression were determined via Western Blot using T cells (CD19⁻TCR β ⁺eYFP⁺) and B cells (CD19⁺TCR β ⁻) sorted from the spleen of MyD88 control and cKO animals. (B) Representative eYFP expression of total splenocytes and iNKT cells (TCR β ⁺CD1d⁺) from MyD88 cKO animals. (C) Representative staining of NKT1, NKT2, and NKT17 lineages (TCR β ⁺CD1d⁺) from the thymus of IL-12R β 2 control and cKO mice, compared to BALB/c control. (D) Representative staining of NKT1, NKT2, and NKT17 lineages (TCR β ⁺CD1d⁺) from the thymus of MyD88 control and cKO mice, compared to BALB/c control. Data are representative of two (A) or at least three (B-D) independent experiments and error bars indicate SEM.

Supplementary Figure 2



Supplementary Figure 2. Loss of IL-12R β 2 and MyD88 signaling in the T cell lineage differentially affect iNKT cell activation following MCMV infection, but not NK cells. Representative flow cytometry of IFN- γ ⁺ NK cells (TCR β ⁻NK1.1⁺) from the spleen and liver of (A) IL-12R β 2 control and cKO mice or (B) MyD88 control and cKO mice at 36 hours post-infection with MCMV. Data are representative of two (A) or three (B) independent experiments.

Supplementary Figure 3



Supplementary Figure 3. NKT1 cells are not detrimentally affected by loss of MyD88 signaling during acute MCMV infection. (A) Representative flow cytometry of NKT1, NKT2, and NKT17 cells ($CD45^+TCR\beta^+CD1d^{tet+}$) in the spleen and liver of MyD88 control and MyD88 cKO animals at 36 hours post-infection with MCMV. NKT cells lineages were differentiated using PLZF, ROR γ t, and T-bet expression. Data are representative of two independent experiments.