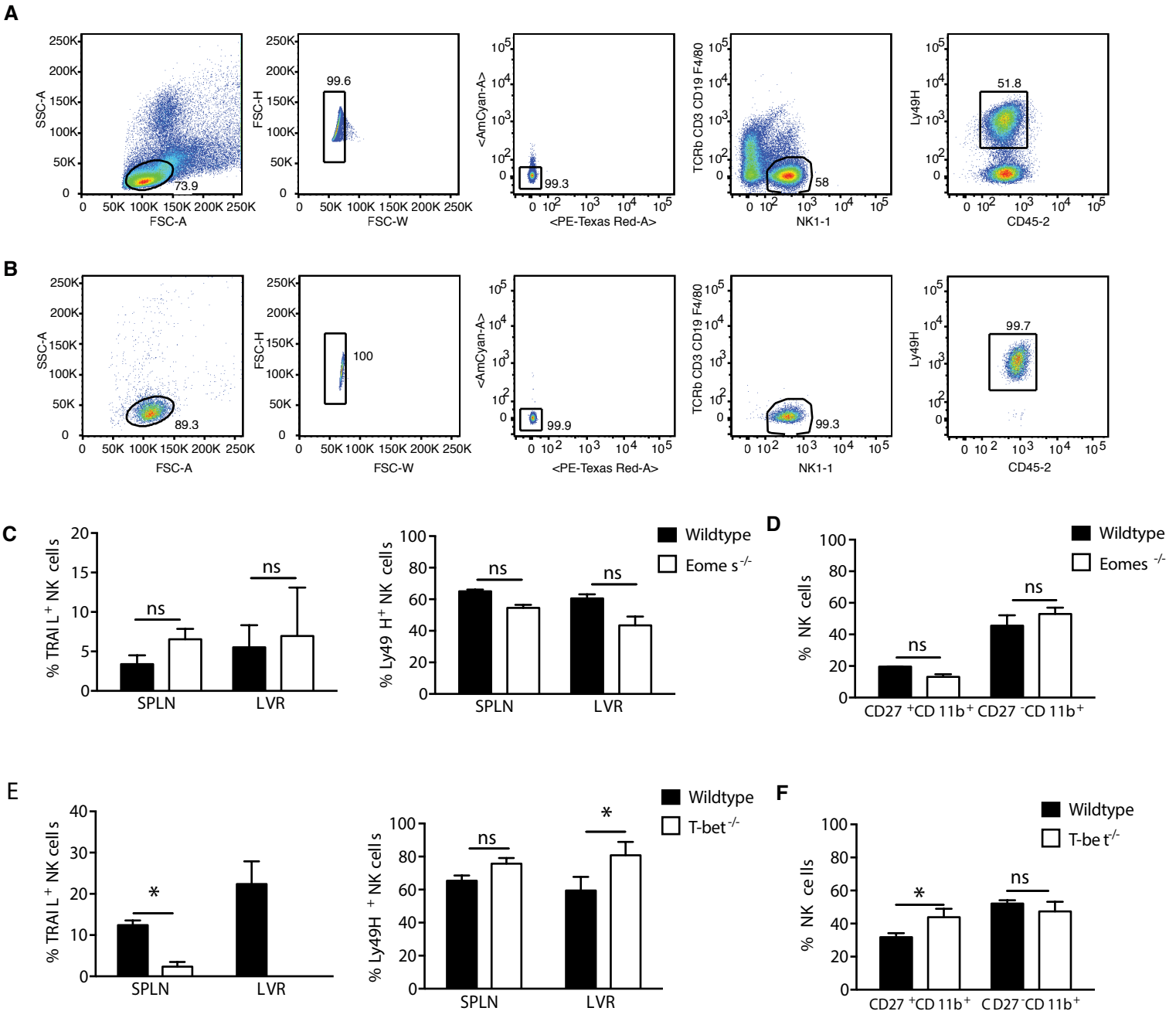
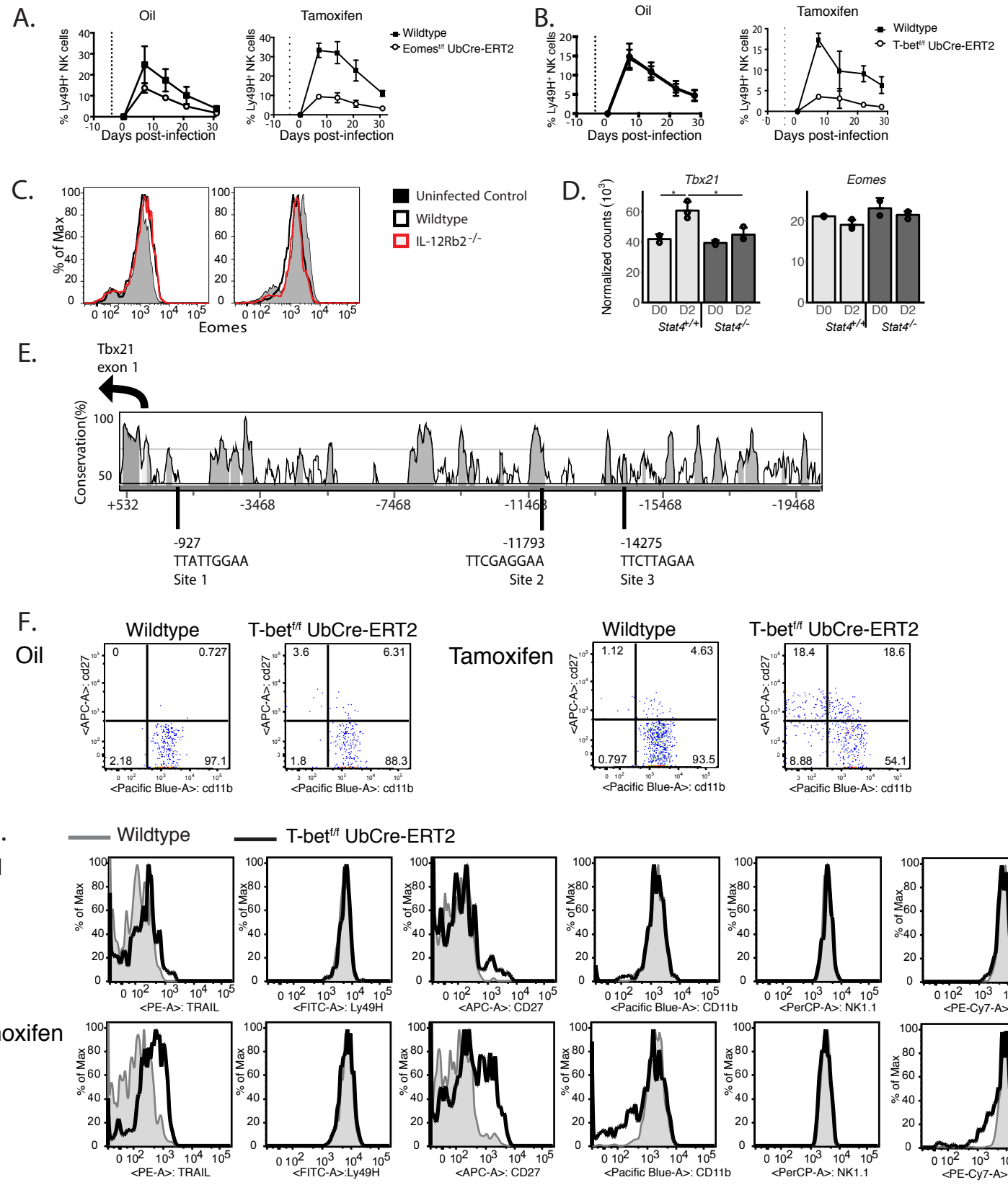


Supplementary Figure 1. Phenotypic changes secondary to deletion of Eomes or T-bet in both normal and lymphopenic mice. WT (CD45.1) and (A & B) Eomes $^{f/f}$ UbCre $^{+}$ (CD45.2) or (C & D) T-bet $^{f/f}$ UbCre $^{+}$ (CD45.2) NK cells were co-transferred into WT (CD45.1x2) mice and treated with tamoxifen or oil at day 0 PT. Assessment of phenotypic markers nine days post transfer for the spleen and liver (A & C) or only spleen (B & D) are shown. Data are representative of two experiments with at least 3 mice in each condition. * $p < 0.05$ and ns, not significant, paired Student t-test.



Supplementary Figure 2. OIl treated floxed NK cells expand comparably to WT counterparts in response to MCMV infection. WT (CD45.1) and Eomesf/f UbCre+ (CD45.2, A) or T-betf/f UbCre+ (CD45.2, B) NK cells were co-transferred into Ly49h-/- mice. Mice were treated with oil or tamoxifen at day -4, -3, and -1 PI and infected with MCMV at D0 PI. Percentages of each population in blood are shown for the indicated time points. (C) Expression of Eomes in WT:IL-12rb2-/- bone marrow chimeric mice at day 2 following infection with MCMV. (D) Normalized counts of Tbx21 and Eomes from RNA-Seq of NK cells from WT:STAT4-/- bone marrow chimeric mice following infection with MCMV. (E) Vista browser image of mouse Tbx21 promoter and enhancer regions (CNS shown in gray shading), plus three predicted STAT4 binding sites. (F-G) WT (CD45.1) and T-betf/f UbCre+ NK cells were co-transferred into Rag2-/-IL2rg-/- mice, which were infected with MCMV and treated with tamoxifen (or oil alone) at 2 weeks PI. Assesment of phenotypic markers in spleen 7 weeks PI (5 weeks post-tamoxifen).