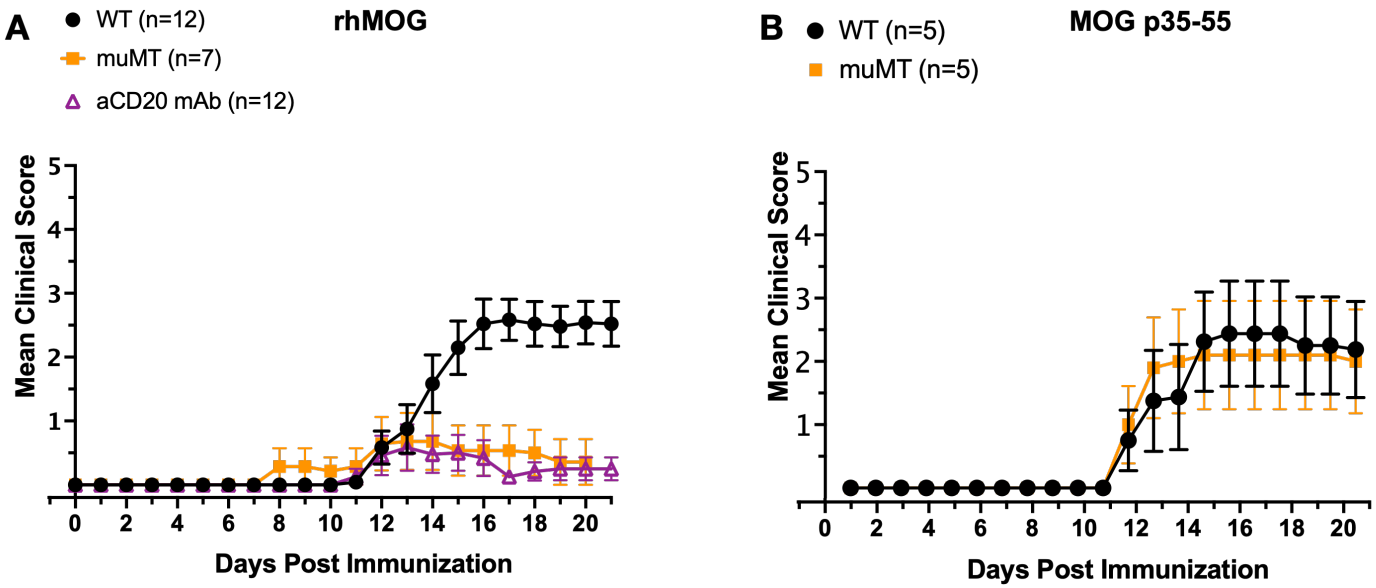
Supplemental Tables:

eTable 1: Full list of differentially expressed genes between anti-CD19 CAR-T cell and control cells versus untransduced activated cells. (Separate Excel Sheet)

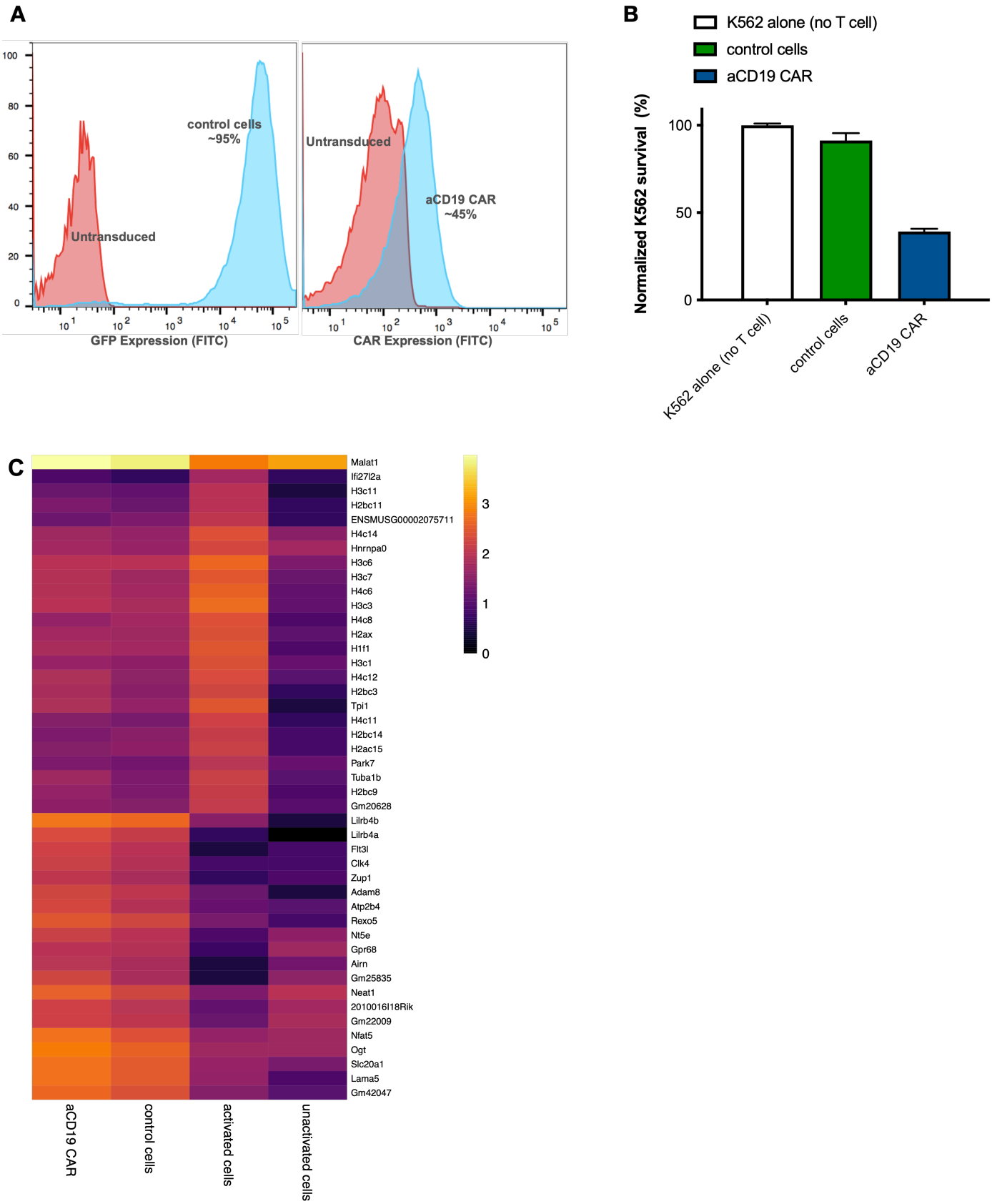
Supplemental Figures:

eFigure 1: rhMOG induced EAE is B-cell dependent



eFigure 1: A. Average EAE Clinical Score after immunization with rhMOG in wild type (WT), muMT (B cell deficient mice), or aCD20 mAb treated mice. **B:** Average EAE Clinical Score after immunization with MOG p35-55 in wild type (WT) or muMT (B cell deficient mice). Shown is average +/- SD.

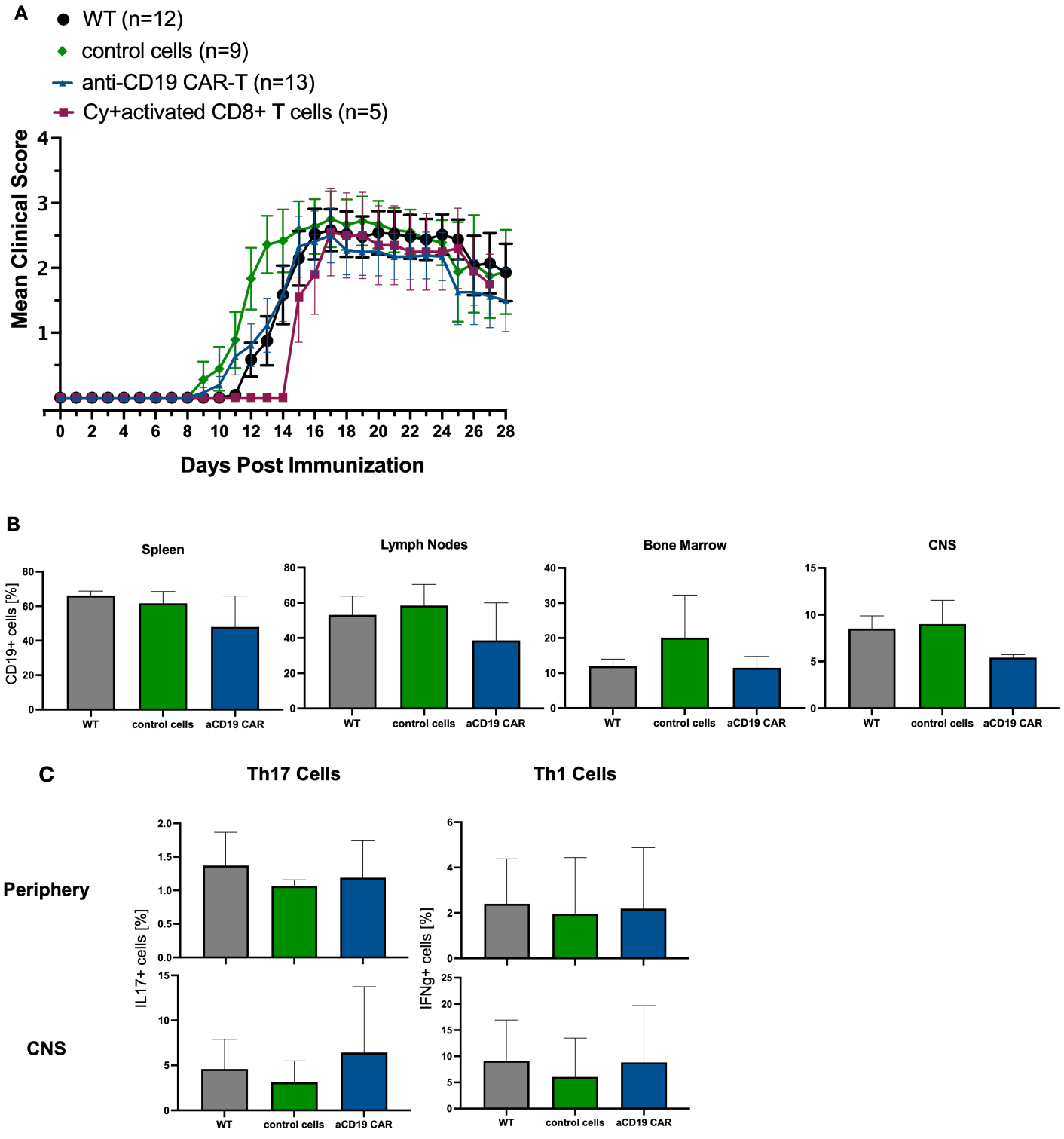
eFigure 2: Characterization of anti-CD19 CAR T cells *in vitro.*

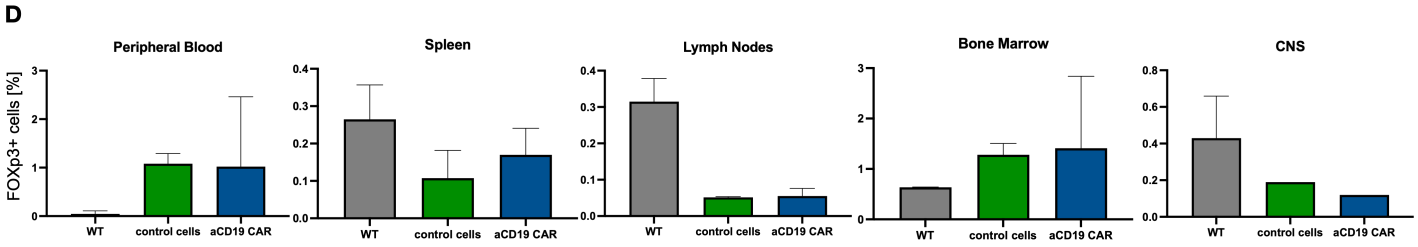


eFigure 2: A. Transfection efficiency of CD8+ mouse T cells of control cells (overexpressing GFP, left) and aCD19 CAR (using CAR specific FITC+ antibody, right) **B.** *In vitro* cytotoxic assay of K562 cells expressing

mouse CD19 on their surface incubated for 48 hours without T cells (K562 alone), control cells, or aCD19 CAR. Shown is average +/- SD. **C.** Heat map of differentially expressed genes between aCD19 CAR and control cells compared to untransduced activated cells alone from bulk RNAseq data looking at expressed genes of treatment cells on day of treatment.

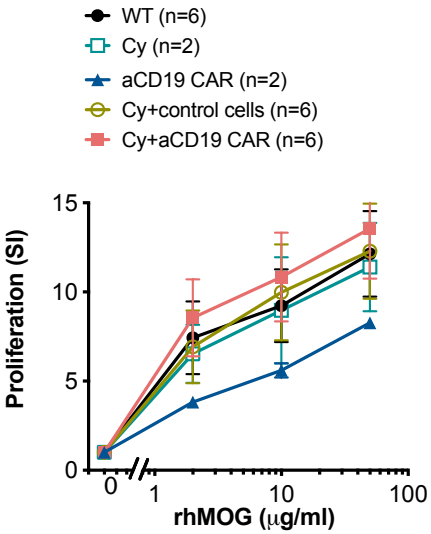
eFigure 3: Effect of anti-CD19 CAR-T cells without pre-treatment on rhMOG-induced EAE





eFigure 3: A. Average EAE Clinical Score after immunization with rhMOG. Shown is average +/- SD. **B.** Frequency of CD45+CD11b-CD19+ cells, average of 2 mouse per disease cohort per compartment which included spleen, inguinal lymph node, and femoral bone marrow, and CNS (brain and spinal cord) on day 28 post immunization. Shown is average +/- SD. **C.** Frequency of CD45+CD4+IL17+ (Th17) or CD45+CD4+IFNg+ (Th1) cells average of 3 mouse per disease cohort from periphery (spleen) or CNS (brain and spinal cord) on day 28 post immunization. **D.** Frequency of CD4+CD25+FOXp3+ cells average of 2 mice per disease cohort per compartment, except CNS which was 1 mouse, on day 28 post immunization. Shown is average +/- SD.

eFigure 4: Absence of proliferative advantage between group in rhMOG induced EAE.



eFigure 4: After immunization of mice with rhMOG for 10 days**,** CD4+ cells from inguinal lymph nodes and spleen were cultured with varying concentrations of rhMOG peptide for 48hrs. Following this, CD4+ cells were observed for thymidine incorporation as a marker for proliferation. Data shown as stimulation index (SI) which is a ratio to the level against background averaged for all the mice/treatment group. Shown is average +/- SD.