

Supplemental Figure 1: Expression of additional markers on ThCTL. Representative histograms of transcription factors (top), NK cell-associated markers (middle), inhibitory, chemokine receptors, and other markers (bottom) on NKG2A/C/E+ or NKG2A/C/Eneg lung CD4 cells from B6 mice 8 dpi. Top: NKG2A/C/E+ CD4+ (dark solid line) or NKG2A/C/Eneg CD4+ (solid gray line) and FMO control (shaded gray). Middle: NKG2A/C/E+ CD4+ (dark solid line), NKG2A/C/Eneg (solid gray line) and CD44^{lo} (dashed line).



Supplemental Figure 2: LCMV induced ThCTL phenotype. Naïve CD4 SMARTA cells were adoptively transferred into congenically marked host mice and then infected with LCMV Armstrong. On 8 dpi, mice were harvested and tissues were isolated to measure the expression of NKG2A/C/E on SMARTA and host CD4 effector cells. (A) Expression of NKG2A/C/E on gated SMARTA or host CD4+CD44^{hi} cells from the indicated organs. mLN = mediastinal lymph nodes (B) Expression of CXCR6 on gated NKG2A/C/E+ (open circle) or NKG2A/C/E negative (closed circle) cells from the indicated organs. (C) Expression of Granzyme B on gated NKG2A/C/E+ (open circle) or NKG2A/C/E negative (closed circle) cells from the indicated organs. (D) SMARTA CD4 effectors were isolated from LCMV infected spleens and flow sorted based on NKG2A/C/E expression. Effectors were then used in an in vitro cytotoxicity assay at a 0.5-0.7 E:T ratio. Blocking anti-MHC-II was added in some wells at 20ug/ml. (A-B) Data are pooled of 2 independent experiments with n=3-5 mice each. Data in (C) is of one experiment with n=3-4 mice. (D) Data are pooled of 2 independent experiments where effectors were sorted from pooled spleens of n=4-5 mice each. Error bars represent SD and significant differences were determined with unpaired two-tailed Student's t-tests where ($\alpha = 0.05$, ** p < 0.005)