

## Figure. S1 In NFIL3 KO mice, α-ASGM1 treatment led to decrease interferon-gamma (IFN-γ) response in HBc-specific CD8 T cells..

(a) Intrahepatic leukocytes (IHLs) were isolated from naïve (Control) and HBV transfected NFIL3 KO mice treated with isotype (Isotype) or anti-ASGM1 (a-ASGM1) for 24 hours on day 10 after hydrodynamic injection with HBV DNA. CD8 T cells of each group from HBV transfected mice were enriched by magnetic beads and mixed with naïve IHL in a ratio of 1:7. Cells were restimulated with HBcAg<sub>18-27</sub> and HBcAg<sub>93-100</sub> peptides for 18 hours. Peptide-activated interferon gamma (IFN- $\gamma$ ) responses were analyzed by a ELISPOT assay. (b) The frequency of peptide-activated IFN- $\gamma$  secretion was quantified as spot-forming cells per 1 × 10<sup>5</sup> cells \*P < 0.05, \*\*P < 0.01, \*\*\*P < 0.001 between selected relevant comparisons, one-tailed unpaired Student's t-test. IHL, intrahepatic leukocytes; SPL, splenocytes.



Figure. S2. ASGM1<sup>+</sup> CD8 T cells expressed more CD44, CD69, CXCR3 and LFA-1 but not other phenotypic markers than ASGM1<sup>-</sup> CD8 T cells.

(a) Intrahepatic leukocytes (IHLs) from naïve NFIL3 KO mice were isolated and a panel of phenotypical markers of ASGM1<sup>+</sup> CD8 T lymphocytes were analyzed by flow cytometry. (b) The comparison of CD44, CD62L, CD69, CD103, CD127, CXCR3 and LFA-1 expression between ASGM1<sup>-</sup> and ASGM1<sup>+</sup> CD8 T cells were analyzed. Data are representative of three independent experiments .



## Figure. S3. The intrahepatic ASGM1<sup>+</sup> LFA-1<sup>hi</sup> CD8 T cells increased after mice were HDI with HBV DNA.

Intrahepatic leukocytes (IHLs) from mice 14 days after HDI with DPBS/HBV DNA were isolated. Cells were stained and analyzed by flow cytometry.



Figure. S4. ASGM1<sup>+</sup>CD44<sup>+</sup>LFA<sup>hi</sup> CD8 T cells were phenotypically liver-resident in not only naïve but also HBV transfected mice.

Intrahepatic leukocytes from naïve or HBV transfected NFIL3 KO mice were isolated and the comparison of CD44, CD69, LFA-1, CXCR3, PD-1, CXCR6, CD49a and CD62L expression between ASGM1<sup>-</sup> and ASGM1<sup>+</sup> CD8 T cells were analyzed.