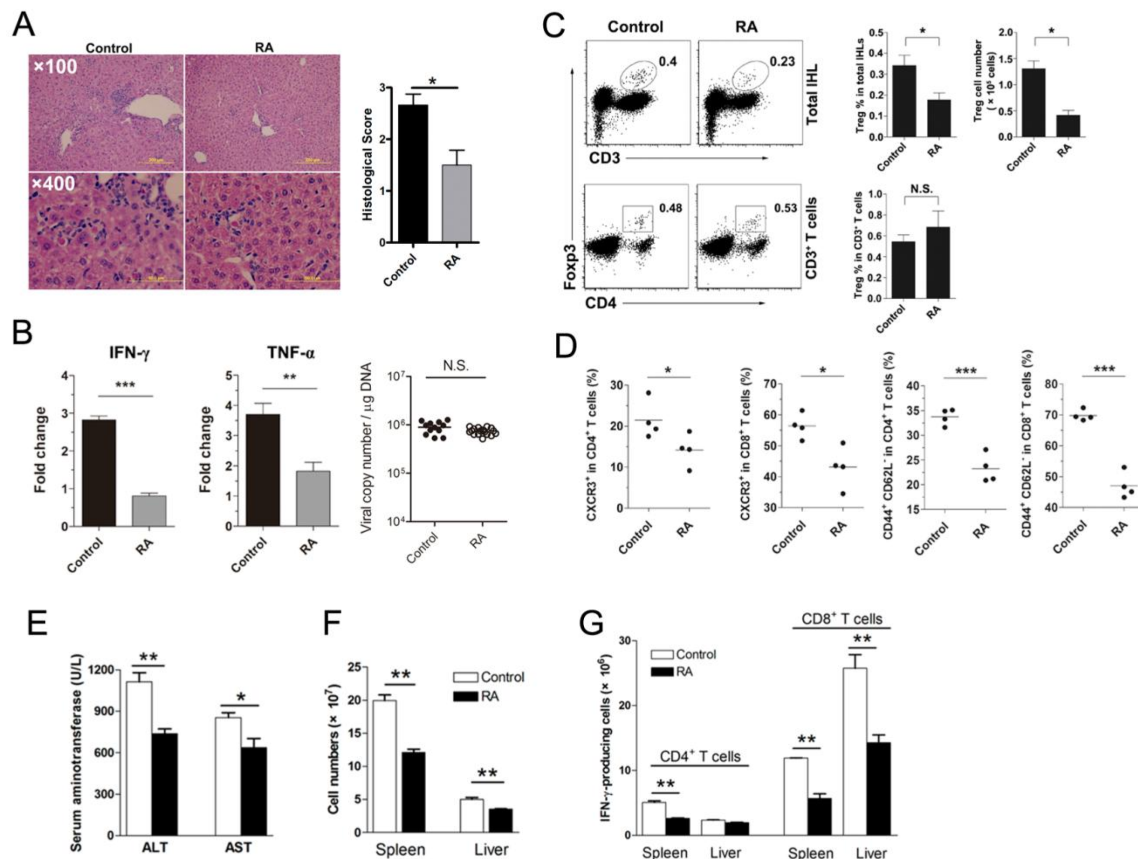

Figure S1. Retinoic acid alleviated liver injury in viral hepatitis.

C57BL/6 mice were injected *i.v.* with 3 $\times 10^9$ pfu of AdLacZ. Mice were injected *i.p.* with 250 μg retinoic acid (RA) or DMSO (control) daily and sacrificed at day 6 post-infection. **(A)** Liver tissues were obtained from Ad-infected control and RA-treated mice, and the tissue sections were stained with H&E. Histological scores were analyzed by three researchers independently. Representative image is shown. **(B)** Intrahepatic lymphocytes were isolated and mRNA levels of cytokines were measured by qPCR. Viral copy numbers in the liver were also analyzed by qPCR. **(C)** Flow cytometric analysis of Foxp3 of intrahepatic IHLs (*upper panel*) and $CD3^+$ T cells (*lower panel*). *Right panel:* Cumulative statistical results of the percentages and absolute cell numbers of $Foxp3^+$ cells in the intrahepatic IHLs or $CD3^+$ T cells, respectively. **(D)** Splenocytes were stained for surface markers (CD3, CD4, CD8, CXCR3, CD44 and CD62L) and examined by flow cytometry. **(E-G)** Infected mice were injected *i.p.* with DMSO (control) or 25 μg RA (moderate dose) daily and sacrificed at 6 days post infection (dpi). **(E)** Serum ALT and AST levels. **(F)** Total numbers of IHLs and splenocytes. **(G)** Absolute numbers of cytokine-producing T cells in the spleen and livers. $*p < 0.05$; $**p < 0.01$.

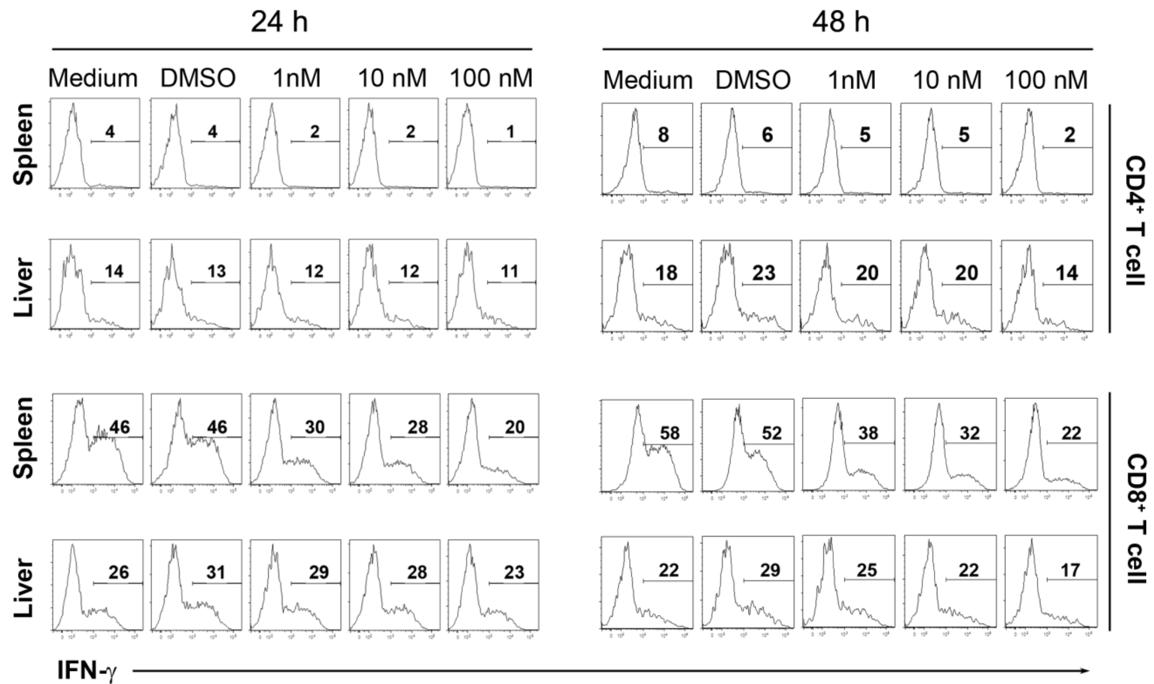

Figure S2. Retinoic acid inhibited IFN-γ production in $CD4^+$ and $CD8^+$ T cells in vitro.

C57BL/6 mice were injected *i.v.* with 3×10^9 pfu of AdLacZ. Splenocytes and IHL were isolated and cultured with RA of indicated concentrations (1, 10 and 100 nM) in the anti-CD3 coated plate. DMSO was used as a solvent control and medium only was used as a blank control. Cells were collected at 24 h and 48 h, and GolgiStop was added at the last 4 h of culture. $CD4^+$ and $CD8^+$ T cells were gated first, follow by analysis of intracellular IFN-γ levels by flow cytometry. Representative images are shown.

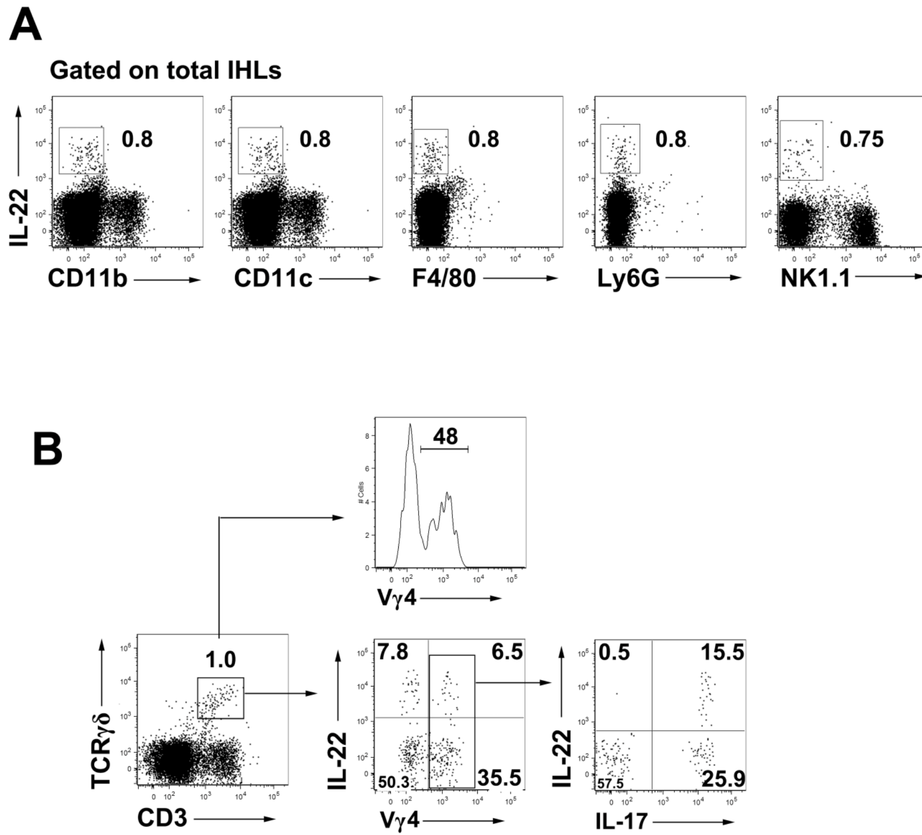

Figure S3. Vγ4 T cells is an important cellular source of IL-22.

IHLs from day 1 infected mice were *in vitro* stimulated for 16 h with recombinant IL-23 (20ng/ml) plus GolgiStop in the last 4 h. Intracellular IL-22 expression was analyzed by flow cytometry. **(A)** IL-22 level was analyzed in CD11b$^+$, CD11c$^+$, F4/80$^+$, Ly6G$^+$, and NK1.1$^+$ cells. **(B)** The IHLs were gated on γδ T cells and Vγ4 T cells were further gated. Vγ4 T cells were analyzed for IL-17 and IL-22 production by flow cytometry. Representative *dot plots* are shown.

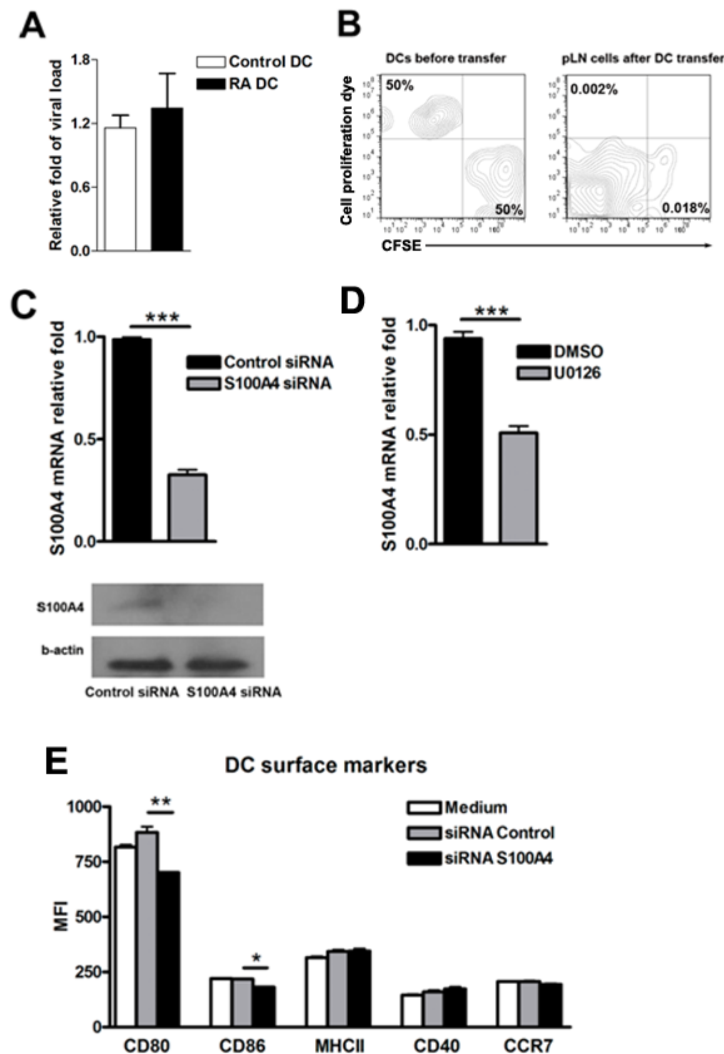

Figure S4. RA-treated DCs have impaired migration ability in vivo and S100A4 siRNA transfection silenced the S100A4 gene and protein expression in DCs.

(A) Control DCs and RA-treated DCs were infected with AdLacZ (MOI: 300) *in vitro* for 24 hours, and the relative fold of viral load was determined by qPCR analysis. **(B)** DCs were infected with AdLacZ (MOI: 300) *in vitro*. Control DCs and RA-treated DCs were labeled with CFSE and cell proliferation dye eflour 670 respectively, and mixed by 1:1 ratio. DCs were injected *s.c.* into the hind foot. Popliteal draining lymph nodes (LNs) were harvested 1 day later and analyzed for DC migration *in vivo*. **(C)** BMDCs were transfected with S100A4 siRNA or control siRNA for 6 h. Cells S100A4 gene and protein levels were detected at 24 h and 72 h by qPCR and Western blot. **(D)** ERK inhibitor U0126 (10 μM) was added into the DC culture system. DMSO was used as a control. Cells were collected at 24 h of cultured for analysis of S100A4 gene levels by qPCR. **(E)** DCs were transfected with S100A4 siRNA or control siRNA for 6 h. DC surface markers were detected at 72 h.