**Table S1. Patient background characteristics for PBMC FACS analysis (Figure 1A and B)**

<table>
<thead>
<tr>
<th>Patient groups</th>
<th>Viral infection</th>
<th>Sex (M/F)</th>
<th>Age (y)</th>
<th>ALT (IU/L)</th>
<th>AST (IU/L)</th>
<th>T-Bil (mg/dL)</th>
<th>ALP (IU/L)</th>
<th>GGTP (IU/L)</th>
<th>PT-INR</th>
<th>PLT (10^9/µL)</th>
<th>IgG (mg/dL)</th>
<th>IgM (mg/dL)</th>
<th>ANA titer&gt;1:80,n (%)</th>
<th>IAIHG scoring#1</th>
<th>Simplified scoring#2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy controls (n=21)</td>
<td></td>
<td>11/10</td>
<td>34.0 [30-55]</td>
<td>15.5 [6-39]</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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</table>

**Table S1B. Patient background characteristics for liver IHC analysis (Figure 1C and D)**

<table>
<thead>
<tr>
<th>Patient groups</th>
<th>Viral infection</th>
<th>Sex (M/F)</th>
<th>Age (y)</th>
<th>ALT (IU/L)</th>
<th>AST (IU/L)</th>
<th>T-Bil (mg/dL)</th>
<th>ALP (IU/L)</th>
<th>GGTP (IU/L)</th>
<th>PT-INR</th>
<th>PLT (10^9/µL)</th>
<th>IgG (mg/dL)</th>
<th>IgM (mg/dL)</th>
<th>ANA titer&gt;1:80,n (%)</th>
<th>IAIHG scoring#1</th>
<th>Simplified scoring#2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver metastasis (Non-tumor part as control) (n=6)</td>
<td></td>
<td>3/3</td>
<td>34 [30-55]</td>
<td>12.5 [8-20]</td>
<td>20 [12-25]</td>
<td>0.6 [0.5-1.9]</td>
<td>238 [161-365]</td>
<td>10 [18-142]</td>
<td>0.96 [0.9-1.13]</td>
<td>20.9 [18.1-27.7]</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

**Supplemental Table 1. Patient background characteristics**

Data are shown as median with ranges in brackets. Notes: #1. Alvarez F et al. J Hepatol 1999, #2. Hennes EM et al. Hepatology 2008. Abbreviations: AIH, autoimmune hepatitis; HAV, hepatitis A virus; HBV, hepatitis B virus; HEV, hepatitis E virus; EBV, Epstein–Barr virus; M, male; F, female; AST, aspartate aminotransferase; ALT, alanine aminotransferase; T-Bil, total bilirubin; ALP, alkaline phosphatase; GGTP, γ-glutamyl transpeptidase; PT-INR, prothrombin time–international ratio; PLT, platelet count; Ig, immunoglobulin; ANA, antinuclear antibody; IAIHG, International Autoimmune Hepatitis Group.
Supplemental Figure 1. The gating strategy of FACS analysis in human PBMC study.
Supplemental Figure 2 Liver pDCs are prone to apoptosis during ConA-induced inflammation.
(A) Representative Annexin V and PI staining and (B) Mean percentages of Annexin V$^+$ cells in liver pDCs. Data represent the mean ± SEM (n=4 per group). **p <0.01 by Student’s t test. Data are representative from two independent experiments.
Supplemental Figure 3. Siglec-H is specifically expressed on pDCs in steady state and inflammatory condition.
Representative Siglec-H histograms of various immune cells in the liver of control or ConA (15 mg/kg, 18h) treated mice. Data are representative from over three independent experiments.
Supplemental Figure 4. The gating strategy of FACS analysis in murine study.
Supplemental Figure 5. Characterization of monocytes/macrophages and neutrophils in pDCs depleted mice in steady state and inflammatory condition.

(A) Representative Ly-6C and CX3CR1 staining of CD45+CD11b+CD11c- gated liver MNCs. CD45+CD11b+CD11c- gated liver MNCs was distinguished as liver macrophages (Ly-6C low CX3CR1-), bone marrow (BM) derived monocytes (Ly-6C high CX3CR1+), and BM derived macrophages (Ly-6C low CX3CR1+). (B) Mean percentages of each cells in CD45+CD11b+CD11c- gated liver MNCs. (C) Representative Ly-6G staining of CD45+ gated liver immune cells. To analyze neutrophils, percoll gradient separation was performed by only 40% percoll. Following centrifugation, immune cells including polymorphonuclear cells were collected at the lower layer, washed, and hemolyzed. (D) Mean percentages of neutrophils in liver CD45+ cells. Data represent the mean ± SEM (n = 4 for the control or control+pDCs-depleted group; n = 5 for the ConA or ConA+pDCs-depleted group). *p < 0.05, **p < 0.01 by Student’s t test. Data are representative from two independent experiments.
Supplemental Figure 6. BM-pDC separation in this study.

Pre-separation
8 days cultured BM cells in Flt-3L

Post-separation
BM-pDCs

B220+microbeads separation

42.2

95.4
Supplemental Figure 7. Adoptive transfer of BM-pDCs at a late stage of disease also ameliorates ConA-induced liver inflammation.

(A) Study design. ConA (15 mg/kg) was intravenously injected into the tail vein of mice. 8 hours later, the mice were intravenously inoculated with Flt-3L-proliferated BM-pDCs (2 × 10⁶ cells/200 µL PBS) or 200 µL PBS alone. All mice were sacrificed and analyzed 18 h after the ConA injection. (B) Serum ALT levels. Data represent the mean ± SEM (n = 6 per group). *p < 0.05 by Student’s t test. Data are combined from two independent experiments.
Supplemental Figure 8. Adoptive transfer of BM-pDCs ameliorates CCl4-induced liver inflammation and DDC-induced cholangitis.

(A) Study design. CCl4 (Wako, Osaka, Japan, 1 ml/kg) in corn oil or corn oil was injected intraperitoneally. One hour later, the mice were inoculated intravenously with BM-pDCs (2 × 10^6 cells/200 µL PBS) or 200 µL PBS alone. All mice were sacrificed and analyzed 20 h after the CCl4 injection.

(B) Serum ALT levels and (C) Serum cytokine concentrations. Data represent the mean ± SEM (n=2 for the control group, n=4 for the CCl4 or CCl4+pDC group). (D) Study design. Mice were freely fed a 0.1% DDC (Sigma-Aldrich, Tokyo, Japan)-enriched or control diet for 7 days. 1 day and 4 days later, the mice were inoculated intravenously with BM-pDCs (2 × 10^6 cells/200 µL PBS) or 200 µL PBS alone. 7 days later, all mice were sacrificed and analyzed. (E) Serum ALT levels, (F) Serum bilirubin, and (G)Th1 (CD45^+TCRβ^+CD4^+IFNγ^+) and Th17 (CD45^+TCRβ^+CD4^+IL-17A^+) in liver CD4 T cells. Data represent the mean ± SEM (n=2 for the control group, n=4 for the DDC or DDC+pDC group). *p <0.05, **p <0.01 by Student’s t test. Data are representative from two independent experiments.
Supplemental Figure 9. Adoptive transfer of Tregs does not ameliorate ConA-induced liver inflammation.

(A) Study design. ConA (15 mg/kg) was intravenously injected into the tail vein of mice. One hour later, the mice were inoculated intravenously with splenic CD4+CD25+Tregs derived from Ly5.1 mice (2 × 10^6 cells/200 µL PBS) or 200 µL PBS alone. All mice were sacrificed and analyzed 18 h after the ConA injection. (B) Representative intracellular Foxp3 and CD25 staining of pre-transferred Tregs. (C) Serum ALT levels. Data represent the mean ± SEM (n=5 per group). (D) Representative CD45.1 and CD4 staining of liver mononuclear cells of Tregs (CD45.1) transferred mice. (E) Cell numbers of transferred pDCs and Tregs in liver during ConA-induced inflammation. (F) Study design. Splenic CD4+CD25+Tregs (2 × 10^6 cells/200 µL PBS), BM-pDCs (2 × 10^6 cells/200 µL PBS), or both pDCs and Tregs (2 × 10^6 cells and 2 × 10^6 cells/200 µL PBS) derived from Ly5.1 mice were intravenously inoculated to Ly5.2 mice. All mice were sacrificed and analyzed 18 h after the ConA injection. (G) Serum ALT levels. (H) Cell numbers of transferred Tregs in each condition. Data represent the mean ± SEM (n=4 per group). **p <0.01 by Student's t test. Data are representative (B and D) or combined (C, E, G, and H) from two independent experiments.
Supplemental Figure 10. Comparison of IL-35 gene expressions among liver pDCs, BM pDCs, and Flt-3L proliferated BM-pDCs. IL-35 genes (IL-12a and Ebi3) expression in natural liver pDCs, natural BM pDCs, and Flt-3L proliferated BM-pDCs. Data represent the mean ± SEM (n = 3 per group). *p < 0.05, **p < 0.01 by ANOVA with Tukey’s multiple comparisons post-hoc test. Data are representative from two independent experiments.
Supplemental Figure 11. IL-10 production and TLR7/9 signaling do not participate in amelioration of ConA-induced inflammation by BM-pDCs.

(A) Study design. ConA (15 mg/kg) or PBS was intravenously injected into the tail vein of mice. One hour later, the mice were inoculated intravenously with WT or IL-10 KO mice derived BM-pDCs (2 × 10^6 cells/200 µL PBS), or 200 µL PBS alone. (B) Serum ALT levels. Data represent the mean ± SEM (n=7 per group). **p <0.01 by ANOVA with Tukey’s multiple comparisons post-hoc test. (C) Study design. ConA (15 mg/kg) or PBS was intravenously injected into the tail vein of mice. One hour later, the mice were inoculated intravenously with WT or MyD88 KO mice derived BM-pDCs (2 × 10^6 cells/200 µL PBS), or 200 µL PBS alone. (D) Serum ALT levels. Data represent the mean ± SEM (n=5 per group). **p <0.01 by ANOVA with Tukey’s multiple comparisons post-hoc test. (E) Study design. WT mice were treated with anti-IL-10R Ab (Biolgend, clone: 1B1.3a) or isotype control (500 µg/head) intraperitonealy 6 h prior to ConA or PBS injection. One hour later, the mice were intravenously inoculated with Flt-3L-proliferated BM-pDCs (2 × 10^6 cells/200 µL PBS) or 200 µL PBS alone. All mice were sacrificed and analyzed 18 h after the ConA injection. (F) Serum ALT levels. Data represent the mean ± SEM (n=4 or 6 per group). Data are combined from two independent experiments.