Supplementary Fig. S1 a **Controls** Anti-CD81 Anti-SR-BI Anti-ApoE – NC PC ···· IgG 500 500 500 500 **Blocking abs PKH67** b P = 0.2504P = 0.0277P = 0.006625 7 25-257 Ø 20 -₩ Ø 15 -C D 33 <sup>↑</sup> Goat-IgG anti-ApoE Rabbit-IgG anti-SR-BI Mouse-IgG + anti-CD81 + **HCV-Exo HCV-Exo HCV-Exo HCV-Exo HCV-Exo HCV-Exo** P = 0.0321P = 0.0090C 207 20-P = 0.060515 -CD33+in PBMCs CD33<sup>+</sup> in PBMCs w B ∃ 10 · 2. C D 33<sup>+</sup>

Goat-IgG

Mouse-IgG

anti-CD81

anti-ApoE

Rabbit-IgG

anti-SR-BI

Supplementary Fig. S1 The role of CD81, SR-BI, and ApoE receptors in HCV-Exo entry and effect on myeloid cells. a Blocking HCV-Exo uptake by myeloid cells. Purified CD33<sup>+</sup> cells were incubated with anti-CD81, SR-BI, and ApoE blocking antibodies or isotype control IgG for 2 h, respectively, and then the PKH67-labelled HCV-Exo were added into the culture for 1 h, after intensive washing, the cells was analyzed by flow cytometry for fluorescence signal. Cells incubated with unlabeled HCV-Exo or PKH67-labelled HCV-Exo alone without blocking antibodies serve as negative control (NC) and positive control (PC). Representative overlaid histograms of the flow data are shown. b, c Functional blocking the effect of HCV-Exo on myeloid cell differentiation. PBMCs were incubated with anti-CD81, SR-BI, and ApoE blocking antibodies or isotype control IgG for 2 h, respectively, and cultured in the presence (B) or absence (C) of HCV-Exo for 3 days. The frequency of CD33<sup>+</sup> myeloid cells in PBMCs were analyzed by flow cytometry.