Supplementary Figure 6. Macrophage Tmem173 knockout attenuates the progression of Atg16l1 knockout-mediated experimental steatohepatitis. (A) Representative H&E staining, oil red O staining, Sirius Red staining, and α-SMA immunohistochemical analysis of liver tissues from MASH mice fed an HFHCD or MCD. (B) NAS, serum ALT levels, and hepatic triglyceride content in the Atg16l1ΔMϕ and Atg16l1ΔMϕTmem173ΔMϕ mice fed an HFHCD or MCD; n=6/group. (C) The gene expression levels of Acta2, Col1a1, and Timp1 in liver tissues from Atg16l1ΔMϕ and Atg16l1ΔMϕTmem173ΔMϕ mice fed an HFHCD or MCD were examined by quantitative real-time PCR; n=6 mice/group. (D) The protein expression levels of α-SMA, collagen-I, and TIMP-1 in liver tissues from Atg16l1ΔMϕ and Atg16l1ΔMϕTmem173ΔMϕ mice fed an HFHCD or MCD were examined by Western blotting. (E) Immunohistochemistry results of F4/80+ cells in liver tissues from MASH mice fed an HFHCD or MCD. (F) The expression of the proinflammatory genes Tnfa, Il6, and Il1b in liver tissues from Atg16l1ΔMϕ and Atg16l1ΔMϕTmem173ΔMϕ MASH mice fed an HFHCD or MCD. (G) Body weights of HFHCD-fed or chow-fed Atg16l1ΔMϕ and Atg16l1ΔMϕTmem173ΔMϕ mice; n=6 per group. (H) The EE of the Atg16l1ΔMϕ and Atg16l1ΔMϕTmem173ΔMϕ mice fed an HFHCD or NCD was calculated as (3.815+1.232×RER)×VO2/lean mass (n=6). ATG16L1, autophagy-related protein 16-like 1; MASH, metabolic dysfunction-associated steatohepatitis; HFHCD, high-fat and high-cholesterol diet; MCD, methionine- and choline-deficient diet; ALT, alanine aminotransferase; AST, aspartate aminotransferase; NAS, NAFLD activity score; EE, energy expenditure. The data are expressed as the mean±SD. *P<0.05, **P<0.01 (unpaired t test or ANOVA).
Supplementary Figure 6. Continued.