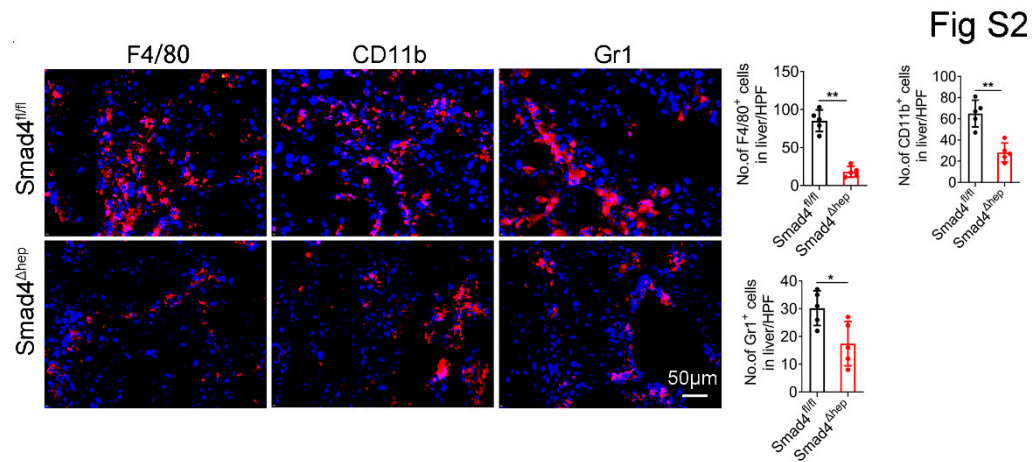


**Figure S1. Detection of Smad4-specific deletion in hepatocytes of Smad4<sup>Δhep</sup> and Smad4<sup>fl/fl</sup> mice.**

(A) Representative immunofluorescence analysis of Smad4 expression in macrophages and myofibroblasts from CCl<sub>4</sub>-treated Smad4<sup>Δhep</sup> and Smad4<sup>fl/fl</sup> mice. F4/80 and α-SMA were used as cell-specific markers (scale bar: 50μm). (B) H&E staining of liver tissues from Smad4<sup>Δhep</sup> and Smad4<sup>fl/fl</sup> mice (scale bars: 100μm, zoom in: 50μm). (C) Sirius Red staining of liver tissues from Smad4<sup>Δhep</sup> and Smad4<sup>fl/fl</sup> mice (scale bars: 100μm, zoom in: 50μm). \*p < 0.05, \*\*p < 0.01.



**Figure S2. Smad4 deficiency in hepatocytes attenuates liver inflammatory response.** Groups of Smad4<sup>fl/fl</sup> and Smad4<sup>Δhep</sup> mice (n = 6 per group) were treated with CCl<sub>4</sub> for 4 weeks to establish the liver fibrosis model. Representative staining and statistical analysis of F4/80, CD11b and Gr1 in liver tissues (scale bar: 50μm). \*p <0.05, \*\*p <0.01.