Supporting Information

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Fig. S1. (*A*) Immune cell infiltration in *macrophage migration inhibitory factor* (*Mif*) WT and *Mif*^{-/-} mice after chronic carbontetrachloride (CCl₄) injury. Overall, the infiltration of NKT cells, T cells, NK cells, and macrophages shows no significant difference between the two strains. (*B*) Immune cell infiltration in *Mif* WT and *Mif*^{-/-} mice after chronic thioacetamide injury. Overall, the infiltration of NKT cells, T cells and NK cells shows no significant difference between the two strains.



Fig. 52. Immune cell infiltration in Cd74 WT and $Cd74^{-/-}$ mice after chronic CCl₄ injury. Overall, the infiltration of NK cells and CD4⁺ cells shows no significant difference between the two strains. Total T cells are as well as CD8⁺ cells are increased in the $Cd74^{-/-}$ mice, but this difference did not reach statistical significance. In contrast the infiltration of the NKT cells was significantly increased in the $Cd74^{-/-}$ mouse strain. *P < 0.05.



Fig. S3. Experimental liver fibrosis in $Cxcr4^{+/-}$ knockout mice after chronic CCl₄ injury. (A) Representative Sirius red staining of WT and $Cxcr4^{+/-}$ mice after challenge with CCl₄. (Magnification: 200×.) (B) No significant difference in $Cxcr4^{+/-}$ mice (n = 13 per group) is validated by the Sirius red-positive area and equal concentrations of hydroxyproline. (C) Expression of fibrosis-related genes in $Cxcr4^{+/-}$ mice. Treatment of $Cxcr4^{+/-}$ with CCl₄ leads to no significant difference mRNAs of *Col1a1*, *Mmp2*, and *Tgfb1* compared with WT mice. Only the mRNA expression of *Timp1* is significantly increased in the heterozygous knockout mice. The infiltration of NK cells and NKT cells shows no significant difference. Only the infiltration of total T cells is slightly, yet not significantly, decreased in the $Cxcr4^{+/-}$ strain, likely the result of a decreased infiltration rate of CD4⁺ cells. *P < 0.05.



Fig. S4. (A) Significantly raised mRNA levels of *Col1a1* in *Cd74^{-/-}* mice treated with CCl₄. mRNA levels of the other fibrosis-related genes *Timp1*, *Mmp2*, and *Tgfb1* show no difference between the $Cd74^{-/-}$ and WT strains under these experimental conditions. (*B*) α -Smooth muscle actin (α -Sma) expression after treatment of mice with recombinant MIF or vehicle concomitantly to CCl₄. Treatment with rMIF resulted in reduced histological α -Sma protein (*Upper*) (magnification: 200×) and α -Sma mRNA expression (*Lower*). Asterisks indicate statistical significance: **P* < 0.05.

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