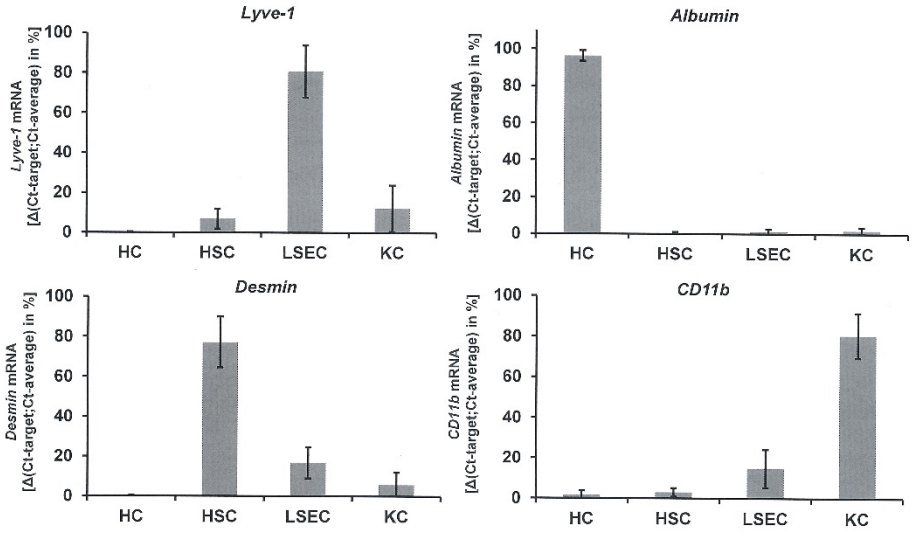
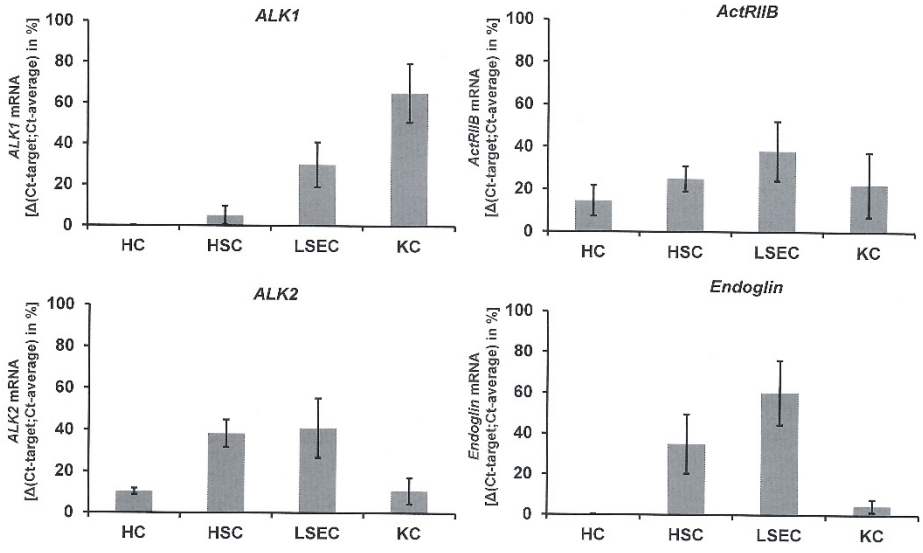
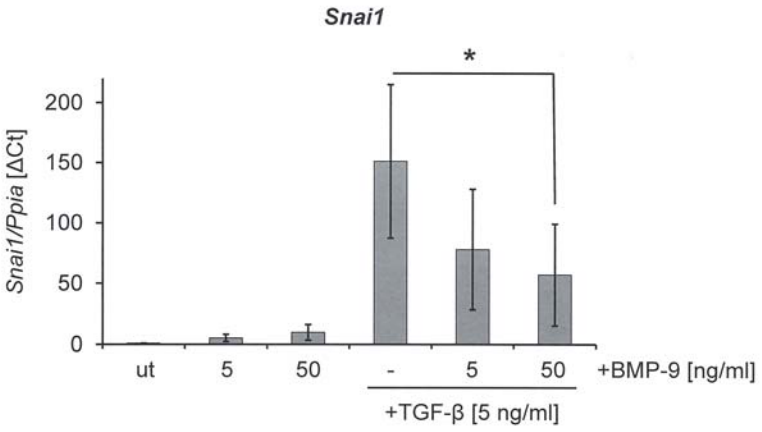


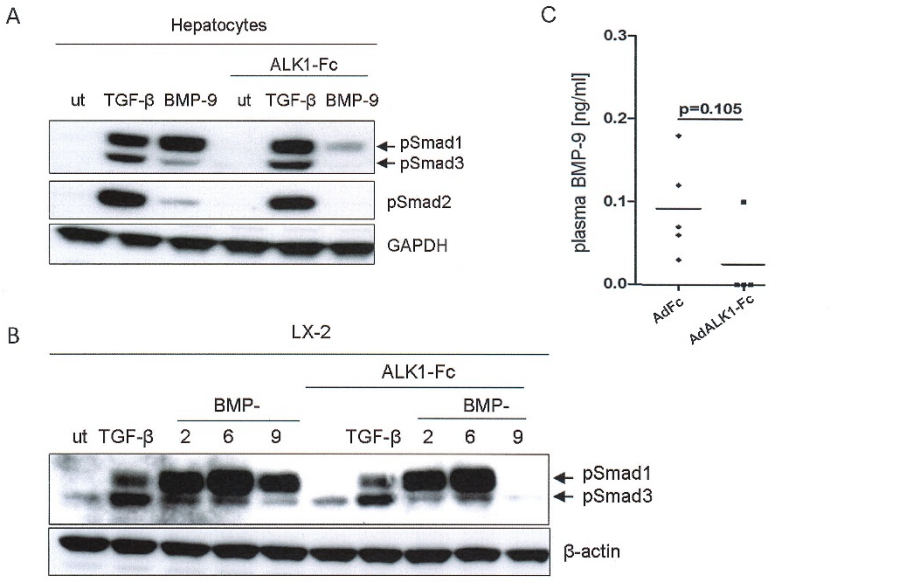
Suppl. fig. 1: Relative purity of the isolated liver cell types. Relative expression levels of mRNA of cell type marker genes (*albumin* for hepatocytes, *Lyve-1* for liver sinusoidal endothelial cells (LSEC), *desmin* for hepatic stellate cells (HSC) and *CD11b* for Kupffer cells (KC)). The average +/- SD of n=8 mice is shown.



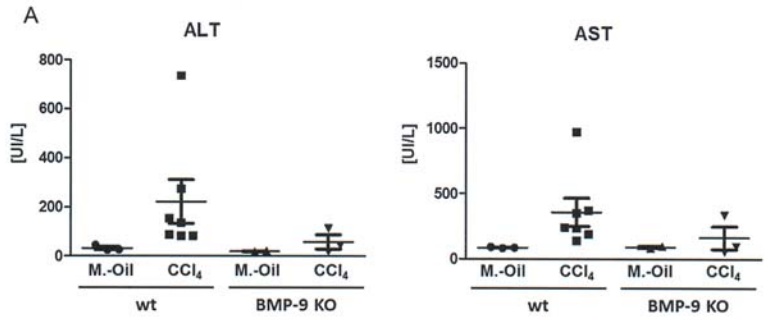
Suppl. fig. 2: Expression of BMP-9 receptors in different liver cell types. Relative expression levels of mRNA of the BMP-type I receptors, *ALK1* and *ALK2*, the high affinity type II receptor, *ActRIIB* and the co-receptor *endoglin* were determined in samples from hepatocytes (HC), liver sinusoidal endothelial cells (LSEC), hepatic stellate cells (HSC) and Kupffer cells (KC) isolated from healthy mouse livers. The average +/- SD of n=3 mice is shown.



Suppl. fig. 3: BMP-9 antagonizes TGF-β mediated induction of Snai1 mRNA. Primary mouse hepatocytes were treated with TGF-β or BMP-9 as indicated for 24h and *Snai1* mRNA-levels were analyzed by real-time PCR.

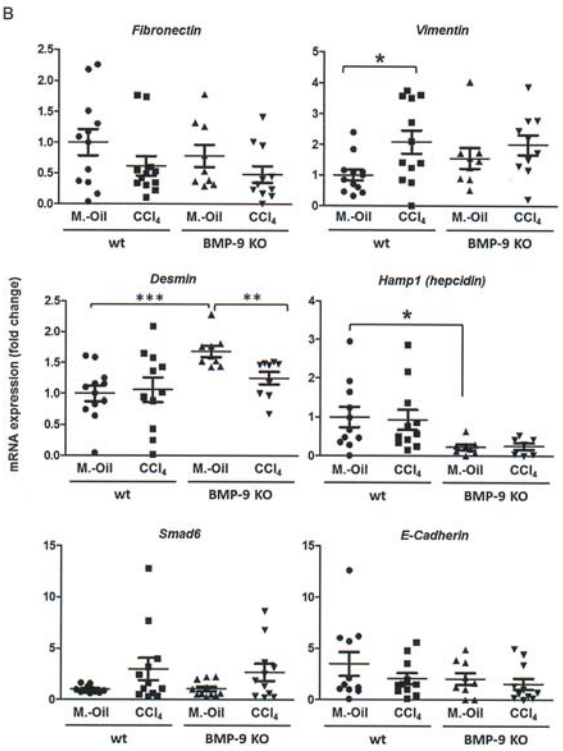


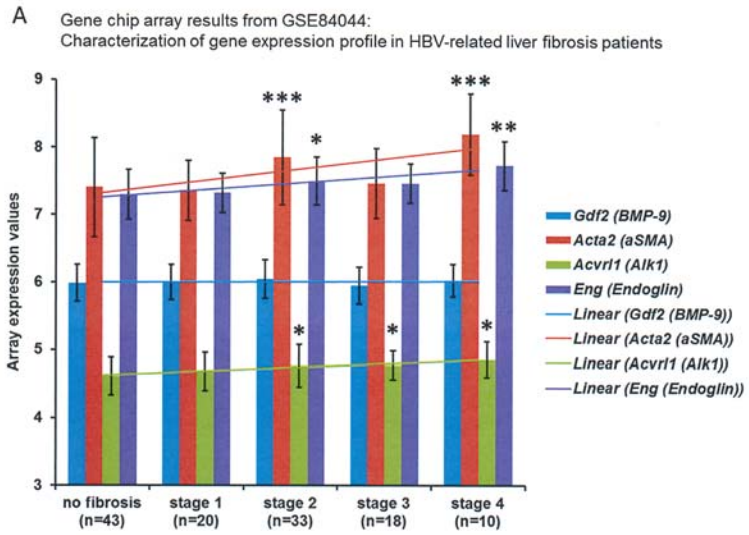
Suppl. fig. 4: ALK1-Fc specifically inhibits BMP-9- but not TGF-β1 or BMP-2 or -6 signalling. A) Primary mouse hepatocytes were stimulated for 1h with TGF-β1 (5 ng/ml) or BMP-9 (5 ng/ml) in the presence or absence of recombinant ALK1-Fc (100 ng/ml) and the phosphorylation status of Smads -1, -2 and -3 was investigated by Western blot. B) LX-2 (a human HSC cell line) were stimulated for 1h with TGF-β1 (5 ng/ml), BMP-9 (5 ng/ml), BMP-2 (20 ng/ml) or BMP-6 (20 ng/ml) in the presence or absence of recombinant ALK1-Fc (100 ng/ml) and the phosphorylation status of Smads -1, -2 and -3 was analyzed as in A). C) Plasma levels of BMP-9 were measured by ELISA in samples from chronic CCl4 treated mice (see figure 5). Free circulating BMP-9 was undetectable in 3 out of 4 AdALK1-Fc injected animals.



Suppl. fig. 5: (A) Levels of the general liver damage markers AST and ALT were measured in the serum of wild type and BMP-9 KO mice as indicated. (B) Expression levels of HSC activation markers (*fibronectin, vimentin, desmin*) as well as direct Smad-1 pathway target genes (*hamp1, Smad6, E-Cadherin*) were investigated by real-time PCR in whole liver samples and were normalized to the house-keeping gene *Gusb*. Data are expressed relative to untreated samples (assigned an arbitrary value of 1) and are mean ± S.E.M. of at least seven animals.

Suppl. fig. 5 (continued)





Suppl. fig. 6: Comparison between *BMP-9, ALK1, Endoglin* and *αSMA* expressions in a (A) human HBV-related liver fibrosis and (B) human HBV-associated acute liver failure (ALF) cohort. Expression levels of the given targets were extracted from published data using "Gene Expression Omnibus" (GEO2r). Averages +/-SD with corresponding linear regressions are shown. Statistic differences in (A) are all related to the "no fibrosis"-group (in (B) they are related to the "normal liver"-group).