Supplemental table 1:

The sequences of siRNA and shRNA used in the experiments.

siRNA	Target sequences
NRP-1(h)	ACGGTCATAGACAGCACCATA
TGF-βRII (h)	TCGGTTAATAACGACATGATA
TGF-βRI (h)	AACAGCATGTGTATAGCTGAA
shRNA	
NRP-1 (h)	CCCTGTTGGTTTCATTTGAATA

Supplemental table 2:

The sequences of primers used in the experiments.

Primers	**Sequences**	**Product length (base pairs)**
β-actin (both for human and mouse)	Forward: GAGACCTTCAACACCCCAGCC Reverse: AATGTCACGCACGATTTCCC	264
Mouse NRP-1	Forward: CATCCACAGCAATTCCACCAAGG Reverse: GGACGTGTCTTGCTGCACAAATC	94
Human TGF-βRII	QT00014350 from Qiagen	108
Human TGF-βRII	QT00083412 from Qiagen	107

Figure legends.

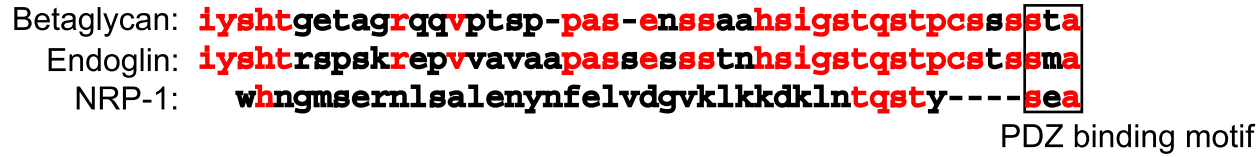
Supplemental Figure 1. Comparison of the intracellular domain of human betaglycan, endoglin, and NRP-1. All of these proteins are well-known for not having any kinase domains but having a PDZ binding motif in the intracellular domain.

Supplemental Figure 2. Isolation of mouse HSC. α-SMA immunostaining was used to confirm the HSCs after 7 days of isolation (A) and the huge upregulation of α-SMA mRNA during the culture activation used for the positive control (B).

Supplemental Figure 3. TGF-β1 downregulated the NRP-1 level in LX2 cells. LX2 cells were serum-starved and treated with 10 ng/mL TGF-β1 for 24 and 48 hours.

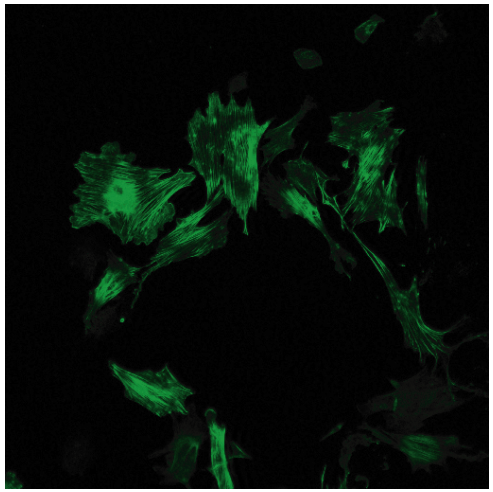
Supplemental Figure 4. The reduced cell proliferation capacity of NRP-1 silenced cells is independent of TGF-β-Smad signaling. LX2 cells were transfected with control and NRP-1 siRNA for 2 days in the present of 5 μM SB431542, and cell proliferation was measured by [3H]-thymidine incorporation assay.

Supplemental Fig 1



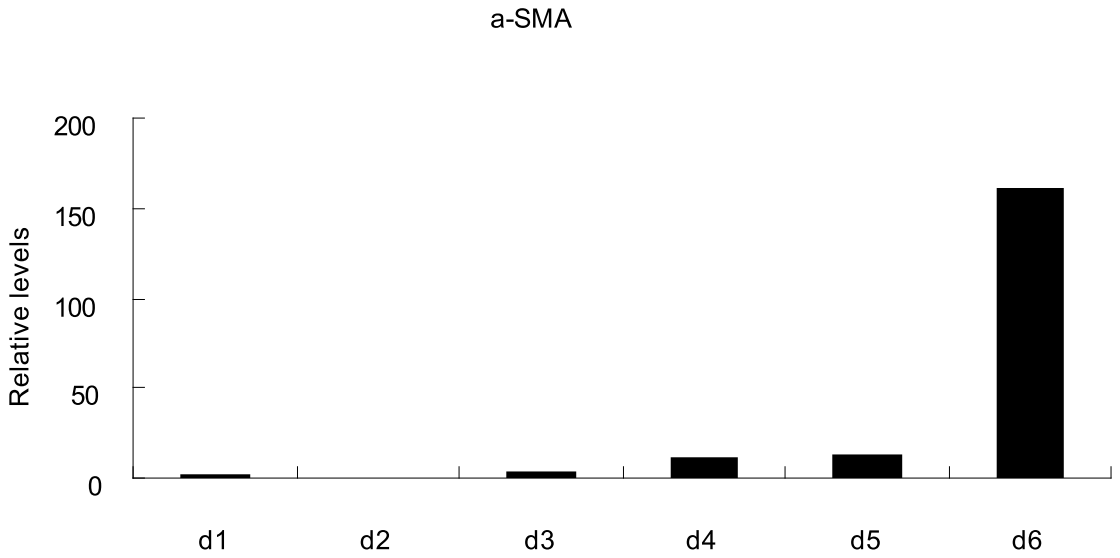
Supplemental Fig 2

A

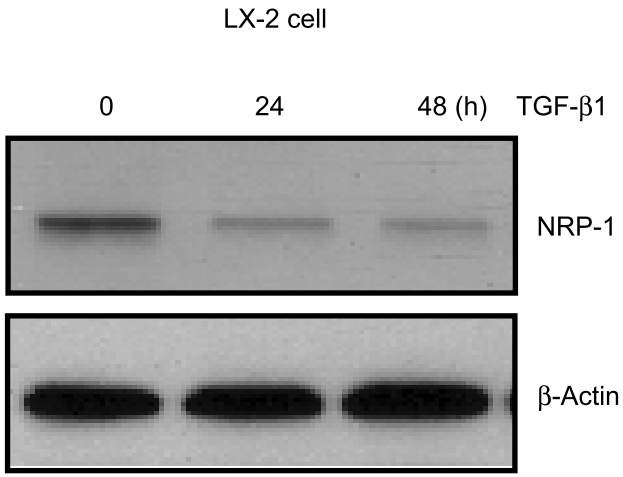


mouse hepatic stellate cell
a-SMA staining

B



Supplemental Fig 3



Supplemental Fig 4