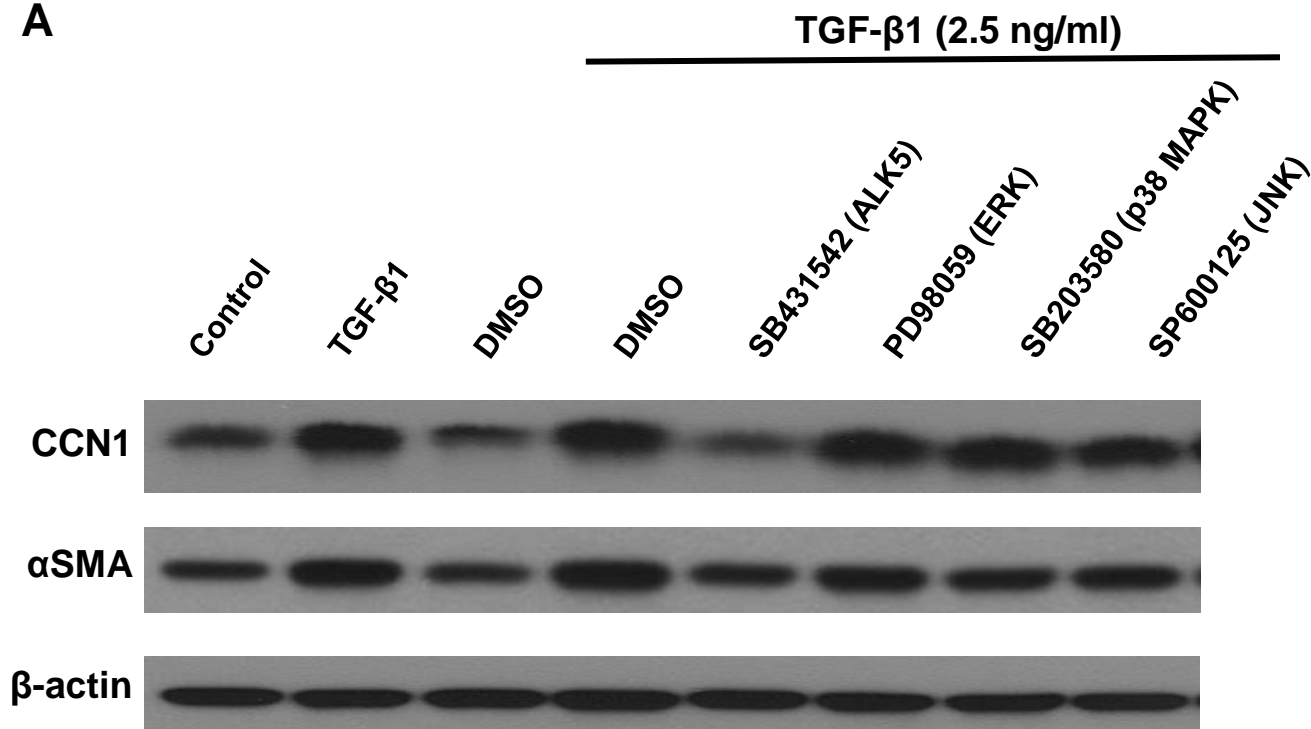
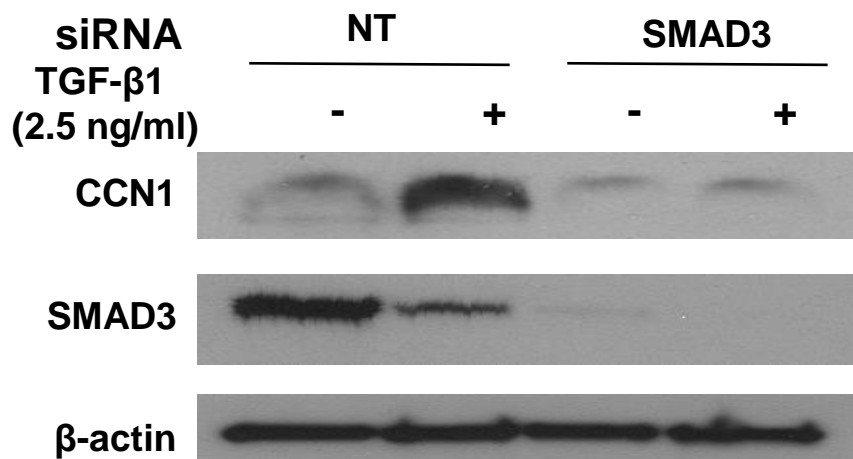


A



B



Supplementary Figure 2: TGF- β 1 mediated CCN1 up-regulation is ALK5/SMAD3-dependent in lung fibroblasts (A) IMR-90 fibroblasts were pre-treated with chemical inhibitors type 1 TGF- β receptor (T β R1/ALK5; SB431542, 1 μ M), extracellular-signal-regulated kinases (ERK; PD98059, 10 μ M), p38 mitogen-activated protein kinases (MAPK; SB203580, 6 μ M), and c-Jun N-terminal protein kinases (JNK; SP600125, 100 nM) for 45 min and then treated with TGF- β 1 (2.5 ng/ml) for 24 h. (B) IMR-90 fibroblasts were transfected with NT and SMAD3 siRNA (100 nM) for 2 days, then serum starved for 24 h and treated with TGF- β 1 (2.5 ng/ml) for 24 h. Expression of CCN1 was evaluated by immunoblotting.