Supporting Information

Wu et al. 10.1073/pnas.0907812106

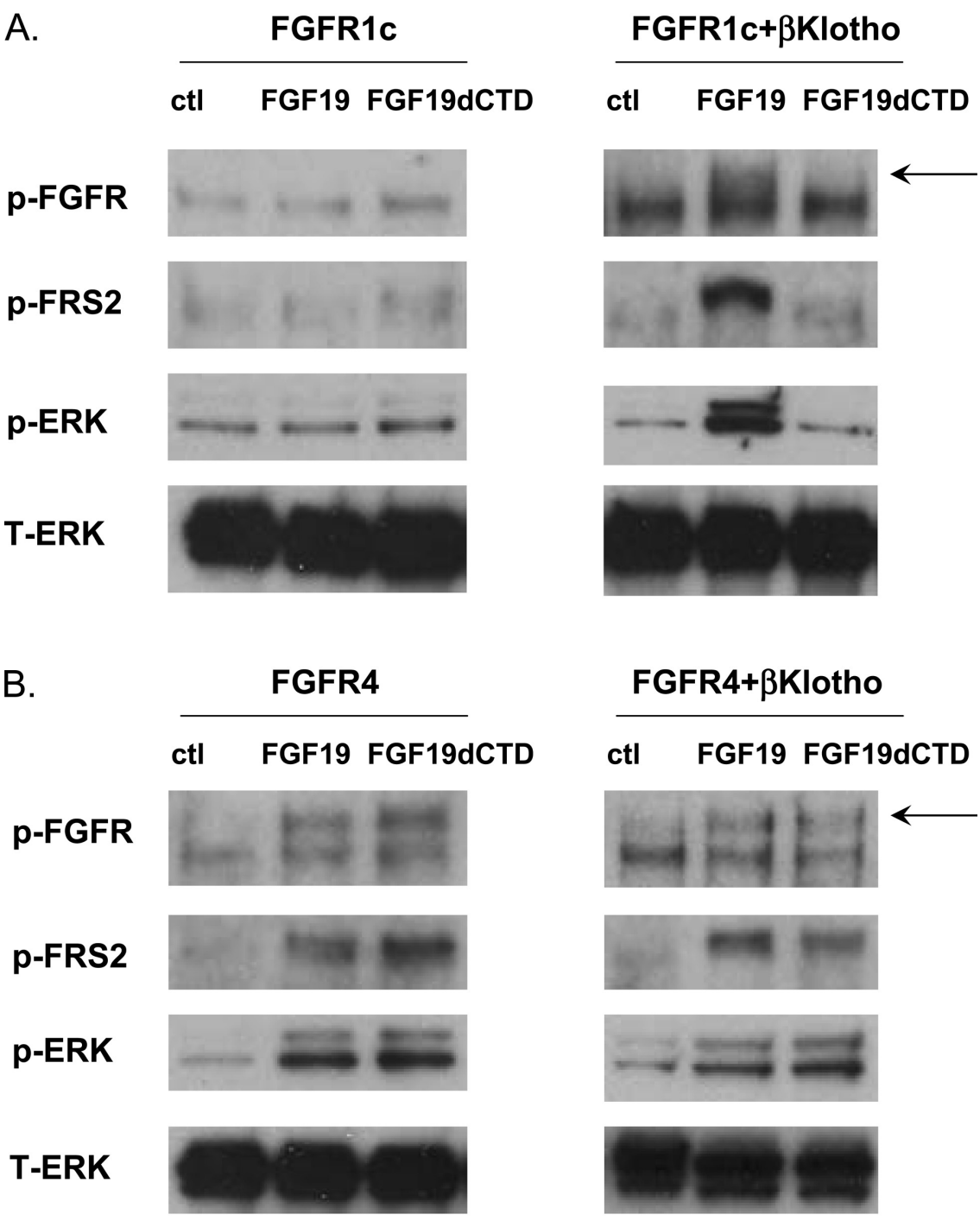

Fig. S1. FGF19 and FGF19dCTD in vitro signaling. L6 cells were co-transfected with expression vectors for FGFR1c or FGFR4 with or without βKlotho. Following overnight serum starvation, cells were stimulated with vehicle or 100 nM recombinant FGF19 or FGF19dCTD for 15 min and snap frozen in liquid nitrogen. Cell lysates were prepared for Western blot analysis using antibodies against phosphorylated FGF receptor (pFGFR), phosphorylated FSR2 (pFRS2), phosphorylated ERK1/2 (pERK), and total ERK1/2 (T-ERK). Antibodies were all purchased from Cell Signaling. Arrow indicates the position of pFGFR.

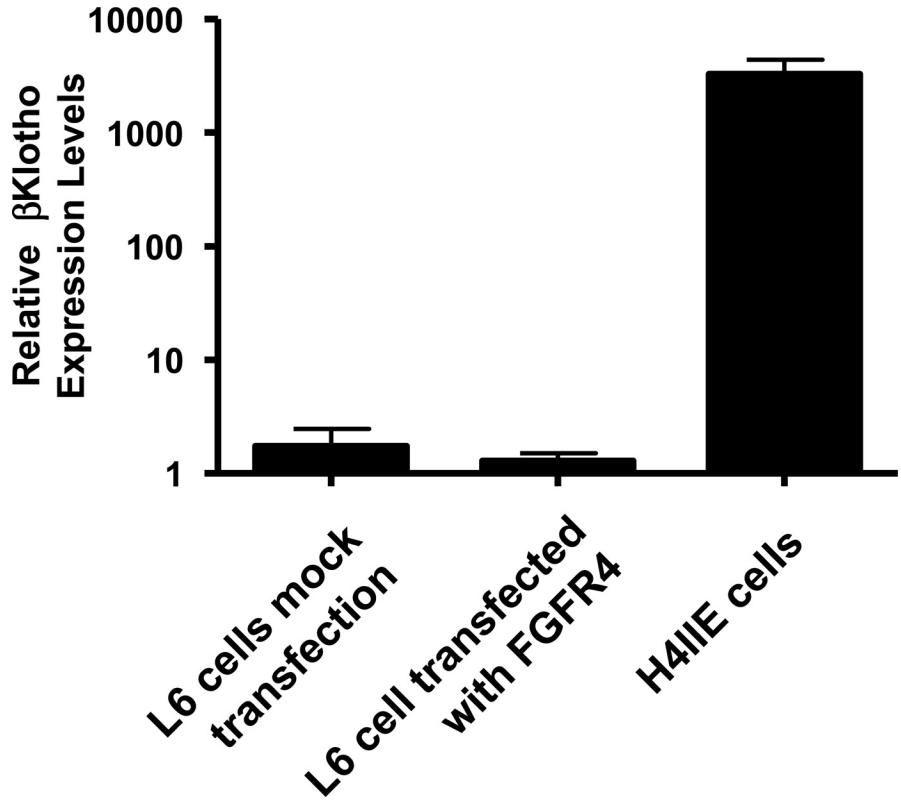

Fig. S2. βKlotho expression level in L6 cells determined by QPCR. L6 cells transfected with an expression vector for FGFR4 or a vector control were serum starved overnight and cells were collected for QPCR analysis. H4IIE, a rat hepatoma cell line that expresses endogenous βKlotho, was used as positive control.

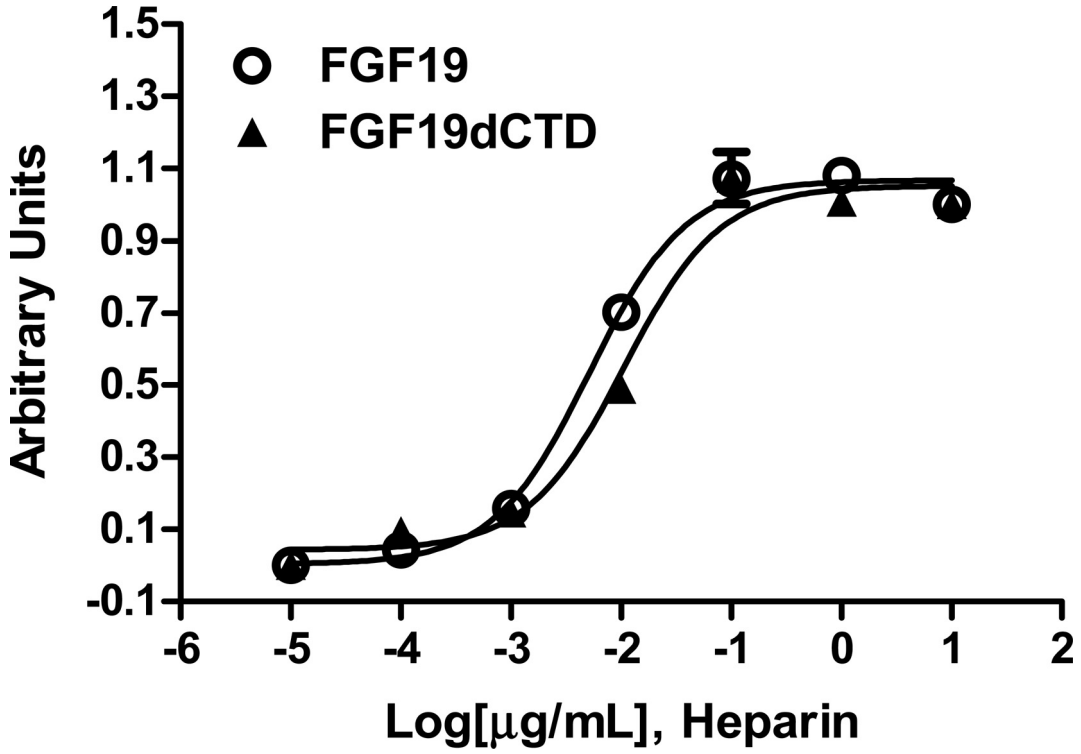

Fig. S3. Solid-phase binding assay measuring the effect of heparin on the interaction between FGFR4 and FGF19 or FGF19dCTD. FGFR4-Fc was captured on the plate, and 200 nM biotinylated FGF19 or FGF19dCTD was added in the presence of various concentrations of heparin. The binding between FGFR4 and FGF19 or FGF19dCTD was detected with a streptavidin-HRP antibody (R&D).

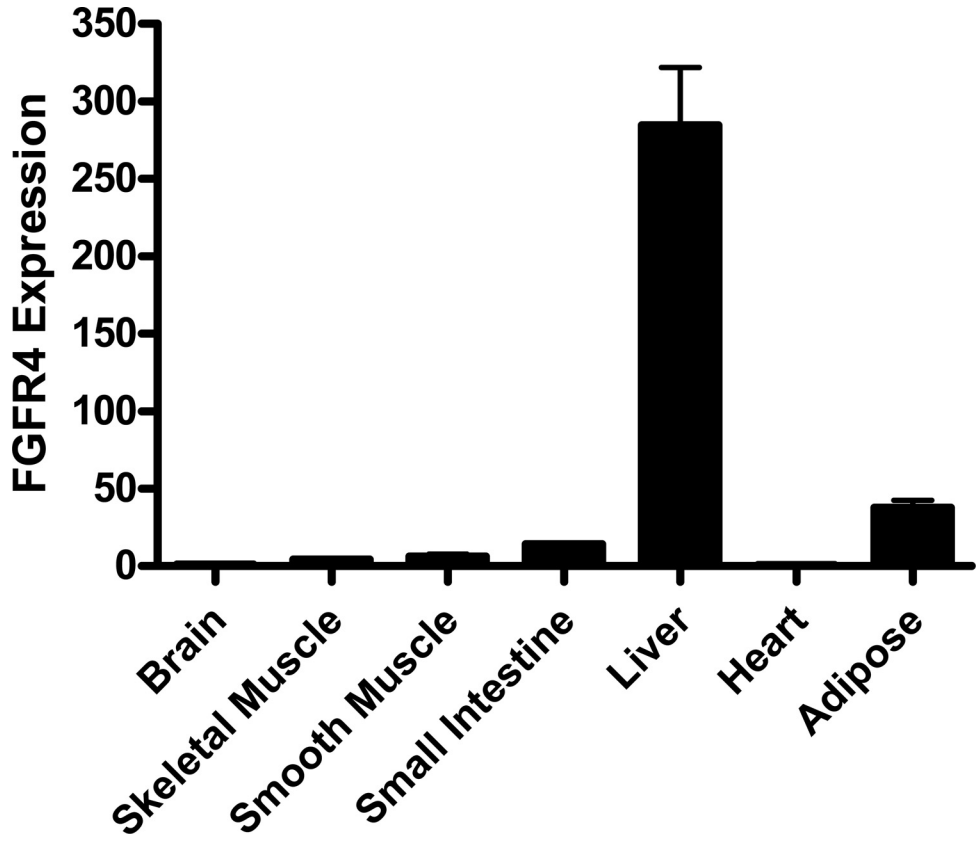

Fig. S4. QPCR analysis of tissue distribution of FGFR4.

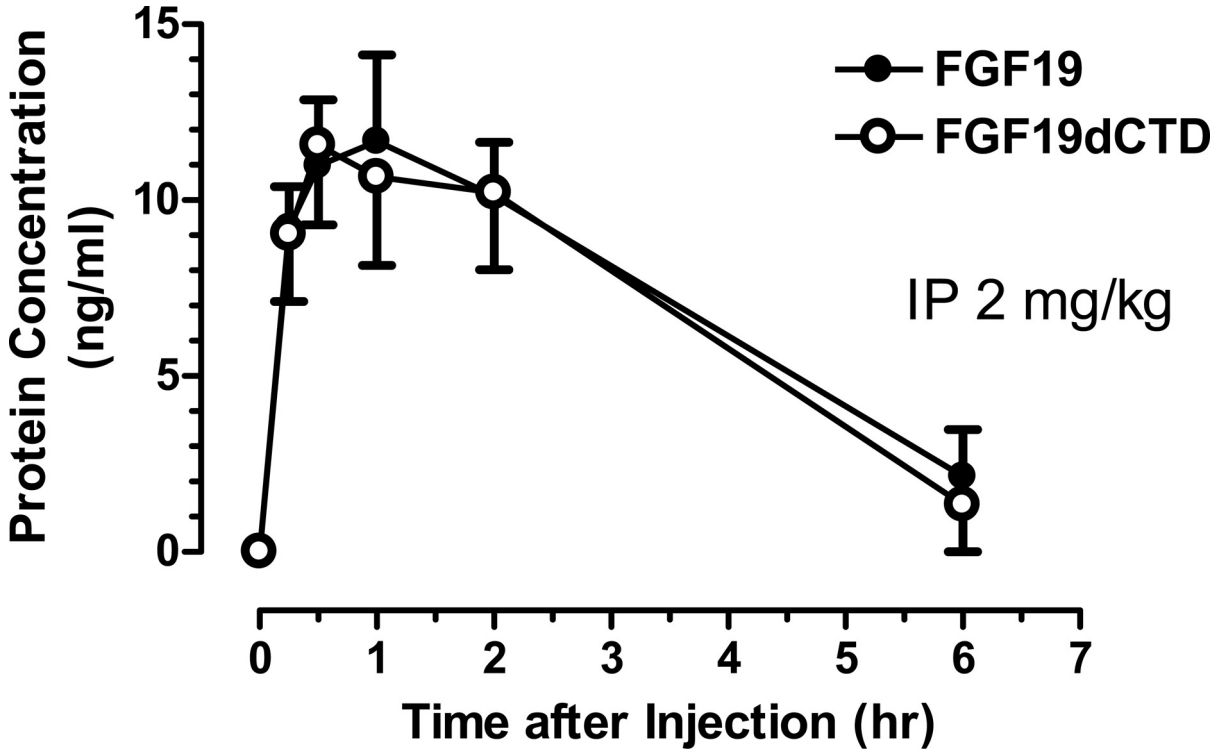

Fig. S5. Pharmacokinetic properties of FGF19 and FGF19dCTD. Following i.p. injections of 2 mg/kg FGF19 or 2 mg/kg FGF19dCTD ($n = 5$), *ob/ob* mouse serum samples were collected 15 min, 30 min, 1 h, 2 h, and 6 h after the injections. FGF19 and FGF19dCTD concentrations were determined by an ELISA format using a polyclonal FGF19 antibody (R&D).