Supplementary Figure S1

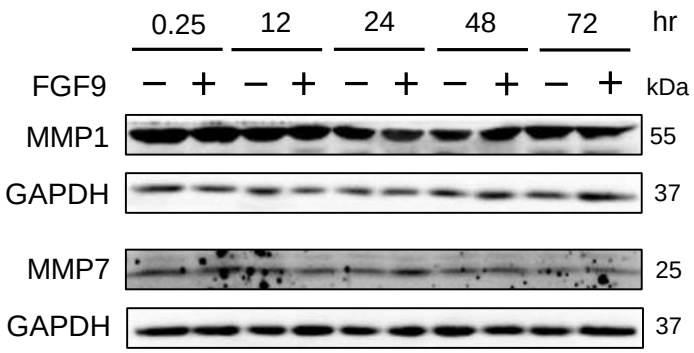

Figure S1. Western blot analysis for protein expression of MMP1 and MMP7 in LLC cells treated with 50 ng/ml FGF9 or BSA for 0.25, 12, 24, 48 and 72 hours, respectively. GAPDH protein expression served as an internal control.

Supplementary Figure S2

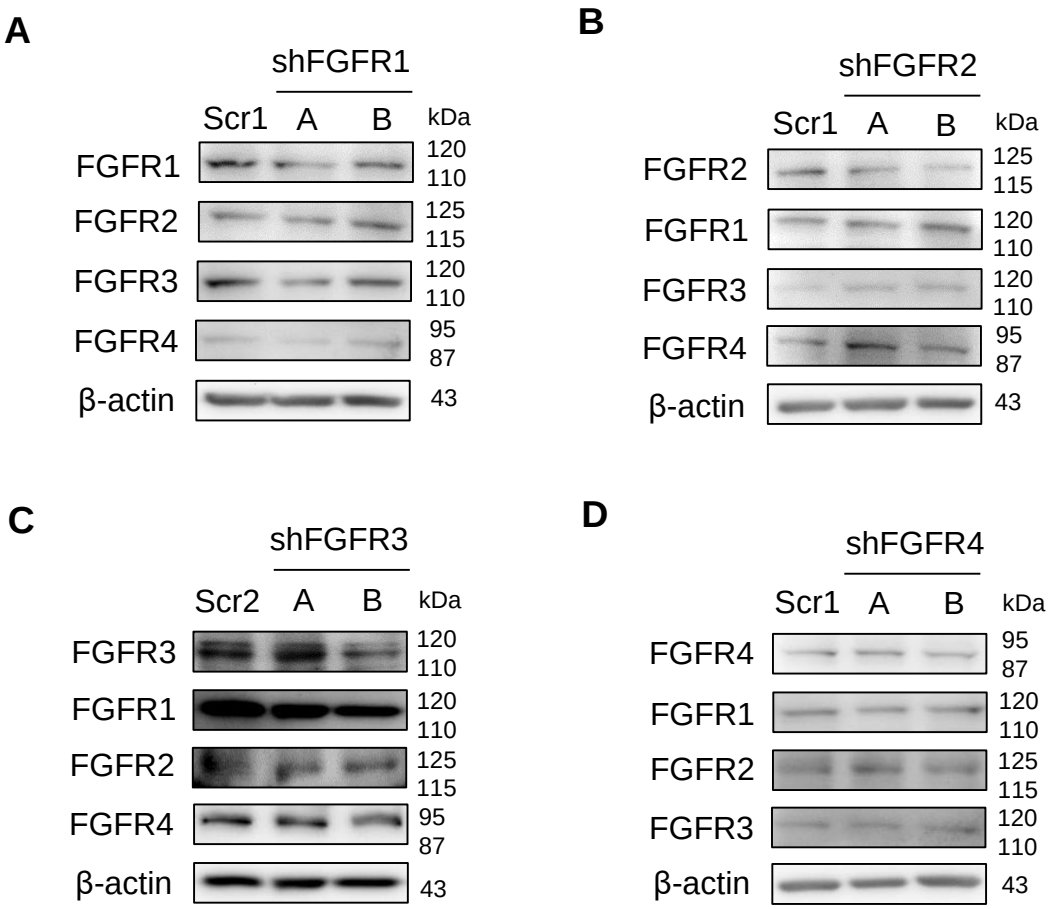

Figure S2. Western blot analysis for gene silencing verification at protein level of FGFR1-4 in LLC cells transfected with a specific shRNA against (**A**) FGFR1, (**B**) FGFR2, (**C**) FGFR3, and (**D**) FGFR4. Two different shRNA targeted sequences were used for each FGF receptor (Table S3). Cells transfected with a non-silencing (scrambled sequence) shRNA in the PLKO.TRC1-puro (Scr1) or PLKO.TRC2-puro (Scr2) vector were used as control as indicated. GAPDH protein expression serves as an internal control.

Supplementary Figure S3

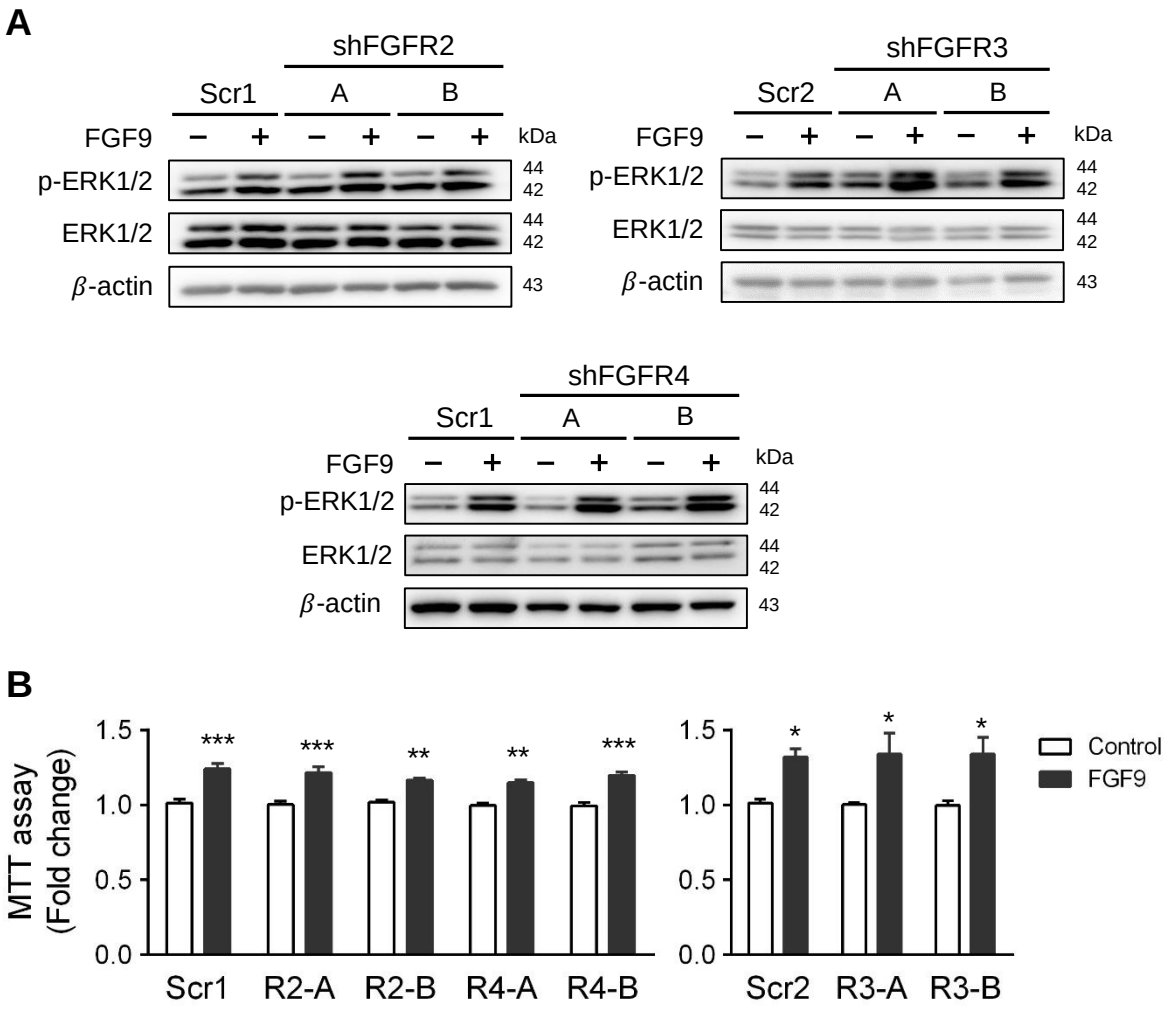

Figure S3. Lentiviral shRNA silencing of FGFR1 inhibited FGF9-induced ERK1/2 phosphorylation and cell proliferation in LLC cells. (**A**) The FGFR2-4 knockdown (shFGFR2, shFGFR3, and shFGFR4) LLC cells and their scrambled control [Scr1 for shFGFR2 and shFGFR4; Scr2 for shFGFR3 (Table S3)] were treated with 0 (Control) or 50 ng/ml FGF9 for 0.25 hour. Levels of ERK1/2 and p-ERK1/2 expressions were analyzed by Western blot assay. (**B**) MTT assay for cell proliferation of shFGFR2, shFGFR3, and shFGFR4 LLC cells treated with 0 (Control) or 50 ng/ml FGF9 or BSA (vehicle control) for 24 hours. All values are represented as the mean ± SEM; n=6. The data were analyzed by two-way ANOVA with Sidak's multiple comparisons post-tests; *p<0.05, **p<0.01 and ***p<0.001 vs. the Control group.