Supplementary Figure and Table Legends

Supplementary Figure S1. FGFR4 silencing in ovarian cancer cells by siRNA transfection. (A) The target, Hs_FGFR4_5, Hs-FGFR4_6, and nontarget scrambled control siRNA sequences used in the transfection experiments. (B), Quantitative real-time PCR analysis of RNAs isolated from the six ovarian cancer cell lines transiently transfected with Hs_FGFR4_5 and Hs-FGFR4_6 and with the nontarget scrambled siRNA for 72 hours, showing significantly lower FGFR4 mRNA expression in cells transfected with the FGFR4 targeting siRNAs ($P < 0.001$). Experiments were repeated in triplicate. (C) Western blot analysis showing downregulation of FGFR4 protein expression in OVCA432 cells transiently transfected with Hs_FGFR4_5 and Hs-FGFR4_6 when compared with the nontarget control siRNA.

Supplementary Figure S2. Effect of FGFR4 overexpression and exogenous FGF1 and on ovarian cancer cell proliferation. The proliferation rates for (A) OVCA429 and (B) OVCA5 cells transduced with the FGFR4 lentiviral construct were significantly higher than were those for mock-transduced control cells ($P < 0.001$). (C) Real-time cell proliferation assay results demonstrating a significant increase in cell proliferation rate when OVCA432 cells were treated with 0.1-1.0 ng/mL FGF1 ($P < 0.001$).

Supplementary Figure S3. Validation of upregulated expression of genes identified via expression profiling of FGFR4-knockdown ovarian cancer cells. The expression of (A) CXCR4 and (B) BNIP3 was significantly increased in OVCA432, OVCA433, and SKOV3 cells with FGFR4 expression knocked down by two different siRNA oligos.
Supplementary Figure S4. Results of immunostaining of ovarian tumor sections obtained from mice. Sections obtained in the (A) FGFR4 trap protein and (B) in vivo FGFR4-targeting siRNA studies were stained with Ki-67. The number of Ki-67–positive cancer cells per unit tumor area in the FGFR4-targeted group was significantly lower than that in the corresponding control group. (C) CXCR4 and (D) BNIP3 protein expression increased significantly in sections obtained from mice injected with the FGFR4 fusion trap protein.

Supplementary Table S1. Detailed protocol used for immunolocalization of FGFR4 in ovarian tumor sections

Supplementary Table S2. Results of Biacore studies of FGFR4mut:Fc FGF-binding affinity. The specificity of FGF ligand binding to FGFR4mut:Fc was assessed using the Biacore T-100 instrument (GE Healthcare), which performed label-free interaction analysis using surface plasmon resonance technology. Protein A or an anti-human IgG antibody (Biacore) was covalently linked with a CM5 chip according to the manufacturer's instructions, and the FGFR4mut:Fc protein was bound to the chip via interaction of the Fc domain with protein-A or the anti-human IgG antibody. FGF ligands were placed in contact with the FGFR fusion protein, also according to the manufacturer's instructions, in the presence of HBS-P buffer (Biacore) supplemented with 50 μg/mL heparin (Sigma). All recombinant FGF ligands were obtained from R&D Systems and PeproTech. The FGF ligands were tested at six to eight concentrations ranging from 4.5 ng/mL to 10.0 μg/mL. The FGF ligands were recombinant and of human origin.
**Supplementary Table S3.** Common differentially expressed genes in both OVCA432 and SKOV3 cells transfected with the FGFR4-specific siRNAs Hs_FGFR4_5 and Hs_FGFR4_6