#12600 Store at -20°C

SignalSilence® FGF Receptor 2 siRNA I

☑ 10 µM in 300 µl (3 nmol)

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For Research Use Only. Not For Use In Diagnostic Procedures.

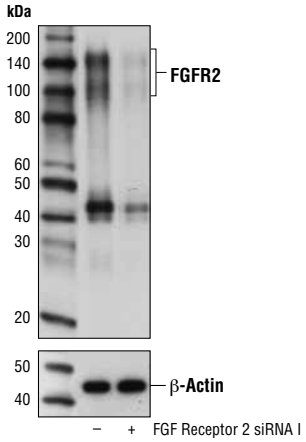
Species Cross-Reactivity: H, (Mk)

Description: SignalSilence® FGF Receptor 2 siRNA I from Cell Signaling Technology (CST) allows the researcher to specifically inhibit FGF Receptor 2 expression using RNA interference, a method whereby gene expression can be selectively silenced through the delivery of double stranded RNA molecules into the cell. All SignalSilence® siRNA products from CST are rigorously tested in-house and have been shown to reduce target protein expression by western analysis.

Background: Fibroblast growth factors (FGFs) produce mitogenic and angiogenic effects in target cells by signaling through cell surface receptor tyrosine kinases. There are four members of the FGF receptor family: FGFR1 (flg), FGFR2 (bek, KGFR), FGFR3, and FGFR4. Each receptor contains an extracellular ligand binding domain, a transmembrane domain, and a cytoplasmic kinase domain (1). Following ligand binding and dimerization, the receptors are phosphorylated at specific tyrosine residues (2). Seven tyrosine residues in the cytoplasmic tail of FGFR1 can be phosphorylated: Tyr463, 583, 585, 653, 654, 730, and 766. Tyr653 and Tyr654 are important for catalytic activity of activated FGFR and are essential for signaling (3). The other phosphorylated tyrosine residues may provide docking sites for downstream signaling components such as Crk and PLCγ (4,5).

FGFR2 has several splicing isoforms with ligand specificity largely determined by alternative splicing of exons 8 (IIIb) and 9 (IIIc). Alternative splicing is cell type specific, resulting in isoforms showing various tissue distribution and biological activities (6,7). Research studies have shown that mutations in the corresponding FGFR2 gene cause syndromes characterized by facial and limb defects, including LADD Syndrome, Crouzon Syndrome, Beare-Stevenson Cutis Gyrata Syndrome, Pfeiffer Syndrome, Apert Syndrome, and Jackson-Weiss Syndrome (8-10). Investigators have also observed mutations and altered expression of FGFR2 in cases of gastric, endometrial, and breast cancer (11).

Specificity/Sensitivity: SignalSilence® FGF Receptor 2 siRNA I inhibits human and monkey FGF Receptor 2 expression.



Western blot analysis of extracts from KATO III cells, transfected with 100 nM SignalSilence® Control siRNA (Unconjugated) #6568 (-) or SignalSilence® FGF Receptor 2 siRNA I (+), using FGF Receptor 2 (D4H9) Rabbit mAb #11835 (upper) or β-Actin (D6A8) Rabbit mAb #8457 (lower). The FGF Receptor 2 (D4H9) Rabbit mAb confirms silencing of FGF Receptor 2 expression, while the β-Actin (D6A8) Rabbit mAb is used as a loading control.

Directions for Use: CST recommends transfection with 100 nM SignalSilence® FGF Receptor 2 siRNA I 48 to 72 hours prior to cell lysis. For transfection procedure, follow protocol provided by the transfection reagent manufacturer. Please feel free to contact CST with any questions on use.

Each vial contains the equivalent of 100 transfections, which corresponds to a final siRNA concentration of 100 nM per transfection in a 24-well plate with a total volume of 300 µl per well.

Quality Control: Oligonucleotide synthesis is monitored base by base through trityl analysis to ensure appropriate coupling efficiency. The oligo is subsequently purified by affinity-solid phase extraction. The annealed RNA duplex is further analyzed by mass spectrometry to verify the exact composition of the duplex. Each lot is compared to the previous lot by mass spectrometry to ensure maximum lot-to-lot consistency.

Entrez-Gene ID #2263
Swiss-Prot Acc. #P21802

Storage: FGF Receptor 2 siRNA I is supplied in RNAse-free water. *Aliquot and store at -20°C.*

Please visit www.cellsignal.com for a complete listing of recommended companion products.

Background References:

(1) Rahman, N. and Stratton, M.R. (1998) *Annu. Rev. Genet.* 32, 95-121.
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(3) Kerr, P. and Ashworth, A. (2001) *Curr. Biol.* 11, R668-R676.
(4) Scully, R. and Livingston, D.M. (2000) *Nature* 408, 429-432.
(5) Tutt, A. and Ashworth, A. (2002) *Trends Mol. Med.* 8, 571-576.
(6) Okada, S. and Ouchi, T. (2003) *J. Biol. Chem.* 278, 2015-2020.
(7) Cortez, D. et al. (1999) *Science* 286, 1162-1166.
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(9) Ouchi, M. et al. (2004) *J. Biol. Chem.* 279, 19643-19648.
(10) Ruffner, H. et al. (1999) *Mol. Cell. Biol.* 19, 4843-4854.
(11) Esashi, F. et al. (2005) *Nature* 434, 598-604.

Applications Key: **W**—Western **IP**—Immunoprecipitation **IHC**—Immunohistochemistry **ChIP**—Chromatin Immunoprecipitation **IF**—Immunofluorescence **F**—Flow cytometry **E-P**—ELISA-Peptide
Species Cross-Reactivity Key: **H**—human **M**—mouse **R**—rat **Hm**—hamster **Mk**—monkey **Mi**—mink **C**—chicken **Dm**—D. melanogaster **X**—Xenopus **Z**—zebrafish **B**—bovine **Dg**—dog **Pg**—pig **Sc**—S. cerevisiae **Ce**—C. elegans **Hr**—Horse **All**—all species expected Species enclosed in parentheses are predicted to react based on 100% homology.