

# SignalSilence® IGF-I Receptor $\beta$ siRNA I (Mouse Specific)

✓ 10 $\mu$ M in 300  $\mu$ l  
(3 nmol)

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

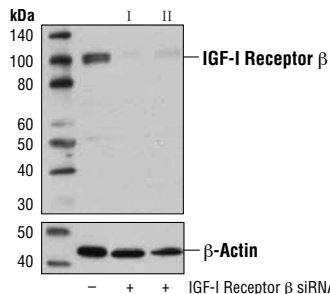
## Species Cross-Reactivity: M

**Description:** SignalSilence® IGF-I Receptor  $\beta$  siRNA I (Mouse Specific) from Cell Signaling Technology (CST) allows the researcher to specifically inhibit IGF-I Receptor  $\beta$  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:** Type I insulin-like growth factor receptor (IGF-IR) is a transmembrane receptor tyrosine kinase that is widely expressed in many cell lines and cell types within fetal and postnatal tissues (1-3). Receptor autophosphorylation follows binding of the IGF-I and IGF-II ligands. Three tyrosine residues within the kinase domain (Tyr1131, Tyr1135, and Tyr1136) are the earliest major autophosphorylation sites (4). Phosphorylation of these three tyrosine residues is necessary for kinase activation (5,6). Insulin receptors (IRs) share significant structural and functional similarity with IGF-I receptors, including the presence of an equivalent tyrosine cluster (Tyr1146/1150/1151) within the kinase domain activation loop. Tyrosine autophosphorylation of IRs is one of the earliest cellular responses to insulin stimulation (7). Autophosphorylation begins with phosphorylation at Tyr1146 and either Tyr1150 or Tyr1151, while full kinase activation requires triple tyrosine phosphorylation (8).

**Directions for Use:** CST recommends transfection with 100 nM SignalSilence® IGF-I Receptor  $\beta$  siRNA I (Mouse Specific) 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  $\mu$ l per well.



Western blot analysis of extracts from NIH/3T3 cells, transfected with 100 nM SignalSilence® Control siRNA (Unconjugated) #6568 (-), SignalSilence® IGF-I Receptor  $\beta$  siRNA I (Mouse Specific) (+), or SignalSilence® IGF-I Receptor  $\beta$  siRNA II (Mouse Specific) #12595 (+), using IGF-I Receptor  $\beta$  (D23H3) XP® Rabbit mAb #9750 (upper) or  $\beta$ -Actin (D6A8) Rabbit mAb #8457 (lower). The IGF-I Receptor  $\beta$  (D23H3) XP® Rabbit mAb confirms silencing of IGF-I receptor  $\beta$  expression, while the  $\beta$ -Actin (D6A8) Rabbit mAb is used as a loading control.

**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 #16001  
Swiss-Prot Acc. #Q60751

**Storage:** IGF-I Receptor  $\beta$  siRNA I (Mouse Specific) is supplied in RNase-free water. Aliquot and store at -20°C.

Please visit [www.cellsignal.com](http://www.cellsignal.com) for a complete listing of recommended companion products.

## Background References:

- (1) Adams, T.E. et al. (2000) *Cell. Mol. Life Sci.* 57, 1050-1093.
- (2) Baserga, R. et al. (2000) *Oncogene* 19, 5574-5581.
- (3) Scheidegger, K.J. et al. (2000) *J. Biol. Chem.* 275, 38921-38928.
- (4) Hernandez-Sanchez, C. et al. (1995) *J. Biol. Chem.* 270, 29176-29181.
- (5) Lopaczynski, W. et al. (2000) *Biochem. Biophys. Res. Commun.* 279, 955-960.
- (6) Baserga, R. et al. (1999) *Exp. Cell Res.* 253, 1-6.
- (7) White, M.F. et al. (1985) *J. Biol. Chem.* 260, 9470-9478.
- (8) White, M.F. et al. (1988) *J. Biol. Chem.* 263, 2969-2980.