

SignalSilence® SirT1 siRNA I



✓ 10 µM in 300 µl (3 nmol)

Orders ■ 877-616-CELL (2355) orders@cellsignal.com
Support ■ 877-678-TECH (8324) info@cellsignal.com
Web ■ www.cellsignal.com

rev. 05/16/16

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

Species Cross-Reactivity: H, (M, R, Mk)

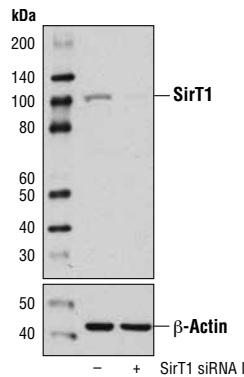
Description: SignalSilence® SirT1 siRNA I from Cell Signaling Technology (CST) allows the researcher to specifically inhibit SirT1 expression using RNA interference, a method whereby gene expression can be selectively silenced through the delivery of double stranded RNA molecules into the cell. All SignalSilence® siRNA products from CST are rigorously tested in-house and have been shown to reduce target protein expression by western analysis.

Background: The Silent Information Regulator (SIR2) family of genes is a highly conserved group of genes that encode nicotinamide adenine dinucleotide (NAD)-dependent protein deacetylases, also known as class III histone deacetylases. The first discovered and best characterized of these genes is *Saccharomyces cerevisiae* SIR2, which is involved in silencing of mating type loci, telomere maintenance, DNA damage response, and cell aging (1). SirT1, the mammalian ortholog of Sir2, is a nuclear protein implicated in the regulation of many cellular processes, including apoptosis, cellular senescence, endocrine signaling, glucose homeostasis, aging, and longevity. Targets of SirT1 include acetylated p53 (2,3), p300 (4), Ku70 (5), forkhead (FoxO) transcription factors (5,6), PPARγ (7), and the PPARγ coactivator-1α (PGC-1α) protein (8). Deacetylation of p53 and FoxO transcription factors represses apoptosis and increases cell survival (2,3,5,6). Deacetylation of PPARγ and PGC-1α regulates the gluconeogenic/glycolytic pathways in the liver and fat mobilization in white adipocytes in response to fasting (7,8). SirT1 deacetylase activity is inhibited by nicotinamide and activated by resveratrol. In addition, SirT1 activity may be regulated by phosphorylation, as it is phosphorylated at Ser27 and Ser47 *in vivo*; however, the function of these phosphorylation sites has not yet been determined (9).

Specificity/Sensitivity: SignalSilence® SirT1 siRNA I inhibits human, mouse, rat, and monkey SirT1 expression.

Directions for Use: CST recommends transfection with 100 nM SignalSilence® SirT1 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.



Western blot analysis of extracts from 293T cells, transfected with 100 nM SignalSilence® Control siRNA (Unconjugated) #6568 (-) or SignalSilence® SirT1 siRNA I (+), using SirT1 (D1D7) Rabbit mAb #9475 (upper) or β-Actin (D6A8) Rabbit mAb #8457 (lower). The SirT1 (D1D7) Rabbit mAb confirms silencing of SirT1 expression, while the β-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 #23411
Swiss-Prot Acc. #Q96EB6

Storage: SirT1 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) Guarente, L. (1999) *Nat. Genet.* 23, 281-285.
- (2) Vaziri, H. et al. (2001) *Cell* 107, 149-159.
- (3) Luo, J. et al. (2001) *Cell* 107, 137-148.
- (4) Bouras, T. et al. (2005) *J. Biol. Chem.* 280, 10264-10276.
- (5) Brunet, A. et al. (2004) *Science* 303, 2011-2015.
- (6) Motta, M.C. et al. (2004) *Cell* 116, 551-563.
- (7) Picard, F. et al. (2004) *Nature* 429, 771-776.
- (8) Rodgers, J.T. et al. (2005) *Nature* 434, 113-118.
- (9) Beausoleil, S.A. et al. (2004) *Proc. Natl. Acad. Sci. USA* 101, 12130-12135.