

SignalSilence® CHD1 siRNA I



✓ 10 µM in 300 µl
(100 transfections)

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

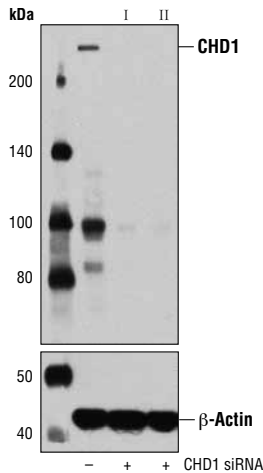
Species Cross-Reactivity: H, Mk

Description: SignalSilence® CHD1 siRNA I from Cell Signaling Technology (CST) allows the researcher to specifically inhibit CHD1 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: Chromodomain-helicase-DNA-binding domain (CHD) proteins have been identified in a variety of organisms (1,2). This family of proteins, which consists of nine members, has been divided into three separate sub-families: subfamily I (CHD1 and CHD2), subfamily II (CHD3 and CHD4), and subfamily III (CHD5, CHD6, CHD7, CHD8 and CHD9). All of the CHD proteins contain two tandem N-terminal chromodomains, a SWI/SNF-related ATPase domain, and a C-terminal DNA binding domain (1,2). The chromodomains facilitate binding to methylated lysine residues of histone proteins and confer interactions with specific regions of chromatin. The SWI/SNF-related ATPase domain utilizes the energy from ATP hydrolysis to modify chromatin structure. CHD1 is a euchromatic protein that associates with the promoters of active genes, and is required for the maintenance of open chromatin and pluripotency in embryonic stem cells (3-6). The two chromodomains of CHD1 facilitate its recruitment to active genes by binding to methyl-lysine 4 of histone H3, a mark associated with transcriptional activation (4-6). Yeast CHD1 is a component of the SAGA and SLIK histone acetyltransferase complexes, and is believed to link histone methylation with histone acetylation during transcriptional activation (6). The CHD2 protein is not well characterized; however, mouse knockout studies suggest important functions in development and tumor suppression. Homozygous CHD2 knockout mice exhibit delayed growth and perinatal lethality (7). Heterozygous knockout mice show increased mortality and gross organ abnormalities, in addition to increased extramedullary hematopoiesis and susceptibility to lymphomas (7,8). CHD2 mutant cells are defective in hematopoietic stem cell differentiation and exhibit aberrant DNA damage responses (8).

Directions for Use: CST recommends transfection with 100 nM SignalSilence® CHD1 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.

Specificity/Sensitivity: SignalSilence® CHD1 siRNA I inhibits human and monkey CHD1 expression.



Western blot analysis of extracts from HeLa cells, transfected with 100 nM SignalSilence® Control siRNA (Unconjugated) #6568 (-), SignalSilence® CHD1 siRNA I (+), or SignalSilence® CHD1 siRNA II #7200 (+), using CHD1 (D8C2) Rabbit mAb #4351 (upper) or β-Actin (D6A8) Rabbit mAb #8457 (lower). The CHD1 (D8C2) Rabbit mAb confirms silencing of CHD1 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 #1105
Swiss-Prot Acc. #014646

Storage: CHD1 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) Hall, J.A. and Georgel, P.T. (2007) *Biochem Cell Biol* 85, 463-76.
- (2) Marfella, C.G. and Imbalzano, A.N. (2007) *Mutat Res* 618, 30-40.
- (3) Gaspar-Maia, A. et al. (2009) *Nature* 460, 863-8.
- (4) Sims, R.J. et al. (2005) *J Biol Chem* 280, 41789-92.
- (5) Flanagan, J.F. et al. (2005) *Nature* 438, 1181-5.
- (6) Pray-Grant, M.G. et al. (2005) *Nature* 433, 434-8.
- (7) Marfella, C.G. et al. (2006) *J Cell Physiol* 209, 162-71.
- (8) Nagarajan, P. et al. (2009) *Oncogene* 28, 1053-62.