

SignalSilence® MDR1/ABCB1 siRNA II



✓ 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. 05/09/16

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

Species Cross-Reactivity: H, (Mk)

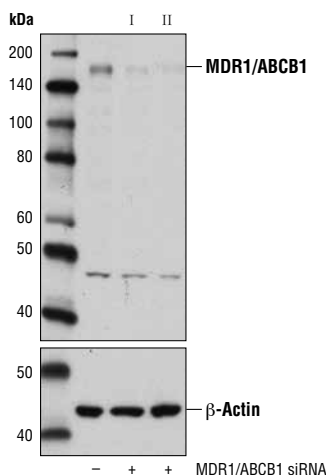
Description: SignalSilence® MDR1/ABCB1 siRNA II from Cell Signaling Technology (CST) allows the researcher to specifically inhibit MDR1/ABCB1 expression using RNA interference, a method whereby gene expression can be selectively silenced through the delivery of double stranded RNA molecules into the 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: MDR1/ABCB1 belongs to the Mdr/Tap subfamily of the ATP binding cassette transporter superfamily (1). MDR1 serves as an efflux pump for xenobiotic compounds with broad substrate specificity. Its substrates include therapeutic agents, such as actinomycin D, etoposide, imatinib, and doxorubicin, as well as endogenous molecules, such as β -amyloids, steroid hormones, lipids, phospholipids, cholesterol, and cytokines (2). Research studies have shown that MDR1 reduces drug accumulation in cancer cells, allowing the development of drug resistance (3-5). On the other hand, MDR1 expressed in the plasma membrane of cells in the blood-brain, blood-cerebral spinal fluid, or blood-placenta barriers restricts the permeability of drugs into these organs from the apical or serosal side (6,7). MDR1 is also expressed in normal tissues with excretory function such as small intestine, liver, and kidney (7). Intracellular MDR1 has been detected in the ER, vesicles, and nuclear envelope, and has been associated with cell trafficking machinery (8). Other reported functions of MDR1 include viral resistance, cytokine trafficking (9,10), and lipid homeostasis in the peripheral and central nervous system (11-13).

Specificity/Sensitivity: SignalSilence® MDR1/ABCB1 siRNA II inhibits human and monkey MDR1/ABCB1 expression.

Directions for Use: CST recommends transfection with 100 nM SignalSilence® MDR1/ABCB1 siRNA II 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 DLD-1 cells, transfected with 100 nM SignalSilence® Control siRNA (Unconjugated) #6568 (-), SignalSilence® MDR1/ABCB1 siRNA I #11879 (+), or SignalSilence® MDR1/ABCB1 siRNA II (+), using MDR1/ABCB1 Antibody #12273 (upper) or β -Actin (D6A8) Rabbit mAb #8457 (lower). The MDR1/ABCB1 Antibody confirms silencing of MDR1/ABCB1 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 #5243
Swiss-Prot Acc. #P08183

Storage: MDR1/ABCB1 siRNA II 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) Furuya, K.N. et al. (1997) *Cancer Res* 57, 3708-16.
- (2) Litman, T. et al. (1997) *Biochim Biophys Acta* 1361, 169-76.
- (3) Chen, C.J. et al. (1986) *Cell* 47, 381-9.
- (4) Kartner, N. et al. (1983) *Cancer Res* 43, 4413-9.
- (5) Chen, G. et al. (1997) *J Biol Chem* 272, 5974-82.
- (6) Brinkmann, U. and Eichelbaum, M. (2001) *Pharmacogenomics J*, 59-64.
- (7) Fromm, M.F. (2004) *Trends Pharmacol Sci* 25, 423-9.
- (8) Miller, D.S. et al. (2008) *Pharmacol Rev* 60, 196-209.
- (9) Ambudkar, S.V. et al. (1999) *Annu Rev Pharmacol Toxicol* 39, 361-98.
- (10) Raviv, Y. et al. (2000) *FASEB J* 14, 511-5.
- (11) Meijer, O.C. et al. (2003) *J Endocrinol* 178, 13-8.
- (12) Karssen, A.M. et al. (2002) *J Endocrinol* 175, 251-60.
- (13) Jeannesson, E. et al. (2009) *Clin Chim Acta* 403, 198-202.