2013 Cell Signaling Technology, Inc.

SignalSilence® c-Rel siRNA II (Mouse Specific)



Orders 877-616-CELL (2355)

orders@cellsignal.com

Support 877-678-TECH (8324)

info@cellsignal.com

Web www.cellsignal.com

New 07/13

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

Species Cross-Reactivity: M

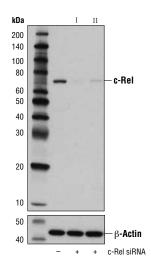
Description: SignalSilence® c-Rel siRNA II (Mouse Specific) from Cell Signaling Technology (CST) allows the researcher to specifically inhibit c-Rel expression using RNA interference, a method whereby gene expression can be selectively silenced through the delivery of double stranded RNA molecules into 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: Transcription factors of the nuclear factor κ B (NF- κ B)/Rel family play a pivotal role in inflammatory and immune responses (1,2). There are five family members in mammals: ReIA, c-ReI, ReIB, NF-kB1 (p105/p50), and NF-κB2 (p100/p52). Both p105 and p100 are proteolytically processed by the proteasome to produce p50 and p52, respectively. Rel proteins bind p50 and p52 to form dimeric complexes that bind DNA and regulate transcription. In unstimulated cells. NF-kB is sequestered in the cytoplasm by IκB inhibitory proteins (3-5). NF-κB-activating agents can induce the phosphorylation of $l_{\kappa}B$ proteins, targeting them for rapid degradation through the ubiquitin-proteasome pathway and releasing NF-κB to enter the nucleus where it regulates gene expression (6-8). NIK and IKK α (IKK1) regulate the phosphorylation and processing of NF- κ B2 (p100) to produce p52, which translocates to the nucleus (9-11).

c-Rel contains an amino-terminal DNA-binding domain referred to as the REL homology domain (REH) and carboxy-terminal transactivation domains. The c-Rel protein is typically inhibited in unstimulated cells by $l\kappa B\alpha$ and $I\kappa B\beta$. c-Rel expression is highest in hematopoietic cells with extensive research studies demonstrating its role in immune cell function and pathogenesis of disease (12,13).

Directions for Use: CST recommends transfection with 100 nM c-Rel siRNA II (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 µl



Western blot analysis of extracts from Neuro-2A cells, transfected with 100 nM SignalSilence® Control siRNA (Unconjugated) #6568 (-), SignalSilence® c-Rel siRNA I (Mouse Specific) #13058 (+) or SignalSilence® c-Rel siRNA II (Mouse Specific) (+), using c-Rel (D4Y6M) Rabbit mAb #12707 (upper) or β-Actin (D6A8) Rabbit mAb #8457 (lower). The c-Rel (D4Y6M) Rabbit mAb confirms silencing of c-Rel 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 #19696 UniProt ID #P15307

Storage: c-Rel siRNA II (Mouse Specific) is supplied in RNAsefree water. Aliquot and store at -20°C.

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

Background References:

- (1) Baeuerle, P.A. and Henkel, T. (1994) Annu Rev Immunol 12,
- (2) Baeuerle, P.A. and Baltimore, D. (1996) Cell 87, 13-20.
- (3) Haskill, S. et al. (1991) Cell 65, 1281-9.
- (4) Thompson, J.E. et al. (1995) Cell 80, 573-82.
- (5) Whiteside, S.T. et al. (1997) EMBO J 16, 1413-26.
- (6) Traenckner, E.B. et al. (1995) EMBO J 14, 2876-83.
- (7) Scherer, D.C. et al. (1995) Proc Natl Acad Sci USA 92, 11259-63.
- (8) Chen, Z.J. et al. (1996) Cell 84, 853-62.
- (9) Senftleben, U. et al. (2001) Science 293, 1495-9.
- (10) Coope, H.J. et al. (2002) EMBO J 21, 5375-85.
- (11) Xiao, G. et al. (2001) Mol Cell 7, 401-9.
- (12) Gilmore, T.D. and Gerondakis, S. (2011) Genes Cancer 2, 695-711.
- (13) Fullard, N. et al. (2012) Int J Biochem Cell Biol 44, 851-60.