SignalSilence® TRAF5 siRNA I



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

Species Cross-Reactivity: H

Description: SignalSilence® TRAF5 siRNA I from Cell Signaling Technology (CST) allows the researcher to specifically inhibit TRAF5 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.

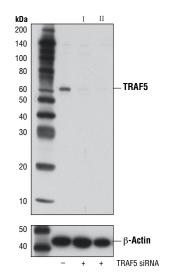
Background: TRAFs (TNF receptor-associated factors) are a family of multifunctional adaptor proteins that bind to surface receptors and recruit additional proteins to form multiprotein signaling complexes capable of promoting cellular responses (1-3). Members of the TRAF family share a common carboxy-terminal "TRAF domain" which mediates interactions with associated proteins; many also contain amino-terminal Zinc/RING finger motifs. The first TRAFs identified, TRAF1 and TRAF2, were found by virtue of their interactions with the cytoplasmic domain of TNF-receptor 2 (TNFRII) (4). The six known TRAFs (TRAF1-6) act as adaptor proteins for a wide range of cell surface receptors and participate in the regulation of cell survival, proliferation, differentiation, and stress responses.

TRAF5 regulates signaling through binding to the cytoplasmic domains of TNFR famly members including CD40, CD27, CD30, OX40, and lymphotoxin- β receptor (5-10). Overexpression of TRAF5 induces NF- κ B activation. Research studies show that cytoplasmic aggregates of TRAF5, as well as TRAF2, are found in Hodgkin-Reed-Sternberg cells, resulting in constitutive NF- κ B activation (11).

Studies of TRAF5 defecient mice suggest that it plays an important role in limiting Th2 immune responses that trigger T-cell mediated inflammatory diseases and asthma (12). Further studies indicate that TRAF5 binds to the IL-6 receptor gp130 and negatively controls Th17 differentation (13). In B-cells, TRAF5 negatively regulates toll-like receptor (TLR) mediated cytokine and antibody production (14).

Directions for Use: CST recommends transfection with 100 nM SignalSilence® TRAF5 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.



New 10/15

Western blot analysis of extracts from A172 cells, transfected with 100 nM SignalSilence® Control siRNA (Unconjugated) #6568 (-), SignalSilence® TRAF5 siRNA I (+), or SignalSilence® TRAF5 siRNA II #60131 using TRAF5 (D3E2R) Rabbit mAb #41658 (upper) or β -Actin (D6A8) Rabbit mAb #8457 (lower). The TRAF5 (D3E2R) Rabbit mAb confirms silencing of TRAF5 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.



Support: +1-978-867-2388 (U.S.) www.cellsignal.com/support

> Orders: 877-616-2355 (U.S.) orders@cellsignal.com

> Entrez-Gene ID #7188 UniProt ID #000463

Storage: SignalSilence[®] siRNA is supplied in RNAse-free water. *Aliquot and store at -20°C*.

For product specific protocols and a complete listing of recommended companion products please see the product web page at www.cellsignal.com

Background References:

- (1) Arch, R.H. et al. (1998) Genes Dev. 12, 2821-2830.
- (2) Chung, J. Y. et al. (2002) J. Cell Sci. 115, 679-688.
- (3) Bradley, J.R. and Pober, J.S. (2001) *Oncogene* 20, 6482-6491.
- (4) Rothe, M. et al. (1994) Cell 78, 681-692.
- (5) Nakano, H. et al. (1996) *J Biol Chem* 271, 14661-4.
 (6) Ishida, T.K. et al. (1996) *Proc Natl Acad Sci USA* 93, 9437-42
- (7) Aizawa, S. et al. (1997) J Biol Chem 272, 2042-5.
- (8) Mizushima, S. et al. (1998) *Gene* 207, 135-40.
- (9) Kawamata, S. et al. (1998) J Biol Chem 273, 5808-14.
- (10) Nakano, H. et al. (1999) *Proc Natl Acad Sci USA* 96, 9803-8.
- (11) Horie, R. et al. (2002) Am J Pathol 160, 1647-54.
- (12) So, T. et al. (2004) J Immunol 172, 4292-7.
- (13) Nagashima, H. et al. (2014) Nat Immunol 15, 449-56.
- (14) Buchta, C.M. and Bishop, G.A. (2014) *J Immunol* 192, 145-50.

Thank you for your recent purchase. If you would like to provide a review visit cellsignal.com/comments.

© 2015 Cell Signaling Technology, Inc.

SignalSilence and Cell Signaling Technology are trademarks of Cell Signaling Technology, Inc.