SignalSilence® RCAS1 siRNA II

#6496 Store at -20°C

☑ 10 µM in 300 µl
(100 transfections)

Cell Signaling TECHNOLOGY®

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rev. 02/11/16

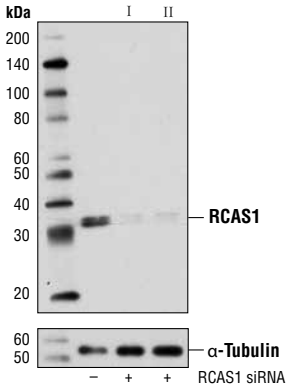
For Research Use Only. Not For Use In Diagnostic Procedures.

Species Cross-Reactivity: H

Description: SignalSilence® RCAS1 siRNA II from Cell Signaling Technology (CST) allows the researcher to specifically inhibit RCAS1 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: Receptor binding cancer antigen expressed on SiSo cells (RCAS1), also called estrogen receptor-binding fragment-associated gene 9 (EBAG9), originally identified as an estrogen-inducible gene (1) was recently found to play a novel role in the adaptive immune response by negatively regulating the cytolytic activity of cytotoxic T-lymphocytes (CTLs) (2). RCAS1 is conserved in phylogeny and is ubiquitously expressed in most human tissues and cells (3,4). There is evidence that tissue expression of RCAS1 is increased in a variety of malignancies, including cancers of the gastro-intestinal tract, liver, lung, breast, ovary, endometrium, and cervix. Moreover, levels of RCAS1 tissue expression are negatively correlated with the prognosis of patients harboring the aforementioned malignancies (4). It is also noteworthy that elevated levels of RCAS1 have been detected in the sera of cancer patients (4). Initial studies indicated that RCAS1 was secreted from cancer cells and functioned as a ligand for a putative receptor expressed on NK cells as well as T- and B-lymphocytes, inducing their apoptosis, which enabled cancer cells to evade immune surveillance (5,6). Subsequent studies have identified RCAS1 as a type-III transmembrane Golgi protein with the ability to regulate vesicle formation, secretion, and protein glycosylation (2,7-9). Indeed, it has been shown that RCAS1 overexpression negatively regulates the cytolytic function of CTLs by negatively regulating protein trafficking from the *trans*-Golgi to secretory lysosomes (2). Furthermore, RCAS1 overexpression delays vesicle transport from the ER to Golgi and causes components of the ER quality control and glycosylation machinery to mislocalize. As a consequence, RCAS1 induces the deposition of tumor-associated glycan antigens on the cell surface, which are thought to contribute to tumor pathogenesis through the mediation of adhesion, invasion, and metastasis (8,9).

Directions for Use: CST recommends transfection with 100 nM RCAS1 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.



Western blot analysis of extracts from HeLa cells, transfected with 100 nM SignalSilence® Control siRNA (Unconjugated) #6568 (-), SignalSilence® RCAS1 siRNA I #6463 (+) or SignalSilence® RCAS1 siRNA II (+), using RCAS1 Antibody #6960 (upper) or α-Tubulin (11H10) Rabbit mAb #2125 (lower). The RCAS1 Antibody confirms silencing of RCAS1 expression, while the α-Tubulin (11H10) 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 #9166
Swiss-Prot Acc. #O00559

Storage: RCAS1 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) Watanabe, T. et al. (1998) *Mol Cell Biol* 18, 442-9.
(2) Rüder, C. et al. (2009) *J Clin Invest* 119, 2184-203.
(3) Tsuchiya, F. et al. (2001) *Biochem Biophys Res Commun* 284, 2-10.
(4) Giaginis, C. et al. (2009) *Histol Histopathol* 24, 761-76.
(5) Matsushima, T. et al. (2001) *Blood* 98, 313-21.
(6) Nakashima, M. et al. (1999) *Nat Med* 5, 938-42.
(7) Reimer, T.A. et al. (2005) *BMC Cancer* 5, 47.
(8) Wolf, J. et al. (2010) *FASEB J* 24, 4000-19.
(9) Engelsberg, A. et al. (2003) *J Biol Chem* 278, 22998-3007.

Applications Key: **W**—Western **IP**—Immunoprecipitation **IHC**—Immunohistochemistry **ChIP**—Chromatin Immunoprecipitation **IF**—Immunofluorescence **F**—Flow cytometry **E-P**—ELISA-Peptide
Species Cross-Reactivity Key: **H**—human **M**—mouse **R**—rat **Hm**—hamster **Mk**—monkey **Mi**—mink **C**—chicken **Dm**—D. melanogaster **X**—Xenopus **Z**—zebrafish **B**—bovine **Dg**—dog **Pg**—pig **Sc**—S. cerevisiae **Ce**—C. elegans **Hr**—Horse **All**—all species expected Species enclosed in parentheses are predicted to react based on 100% homology.