

SignalSilence® ADAM9 siRNA I

✓ 10 µM in 300 µl (3 nmol)

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New 12/12

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

Species Cross-Reactivity: H, (Mk)

Description: SignalSilence® ADAM9 siRNA I from Cell Signaling Technology (CST) allows the researcher to specifically inhibit ADAM9 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.

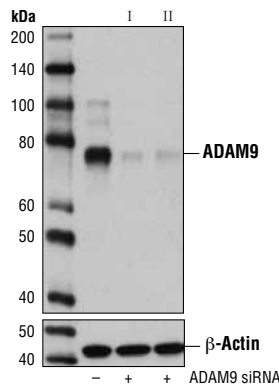
Background: The ADAM (A Disintegrin and A Metalloprotease) family of multidomain membrane proteins influences cell signaling and adhesion by shedding cell surface proteins such as cytokines and growth factors, by influencing cell adhesion to the extracellular matrix (ECM), and by directly remodeling the ECM. Conserved domains in ADAM family members include a prodomain, a zinc-dependent metalloprotease domain, a disintegrin domain, a cysteine-rich domain, an EGF-like sequence, and a short cytoplasmic tail (1,2).

The prodomain is thought to aid in protein folding. Disintegrin and cysteine-rich domains mediate adhesion, at least in part, through binding to integrins. Phosphorylation of the cytoplasmic tail as well as its interaction with other signaling proteins may influence intra- and extracellular signaling (1). ADAM9 is widely distributed and has been shown to affect migration in skin keratinocytes (3,4). Research studies have shown that ADAM9 is overexpressed in prostate cancer (5), pancreatic cancer (6), gastric cancer (7), and has been linked to invasion and metastasis in small cell lung cancer (8). Research has also shown that an alternatively spliced short (50 kDa) form of ADAM9 containing protease activity is involved in tumor cell invasion (9).

Specificity/Sensitivity: SignalSilence® ADAM9 siRNA I inhibits human and monkey ADAM9 expression.

Directions for Use: CST recommends transfection with 100 nM SignalSilence® ADAM9 siRNA I 48 to 72 hours prior to cell lysis. For transfection procedure, follow the 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 HeLa cells, transfected with 100 nM SignalSilence® Control siRNA (Unconjugated) #6568 (-), SignalSilence® ADAM9 siRNA I (+), or SignalSilence® ADAM9 siRNA II #12085 (+), using ADAM9 (D64B5) Rabbit mAb #4151 (upper) or β-Actin (D6A8) Rabbit mAb #8457 (lower). The ADAM9 (D64B5) Rabbit mAb confirms silencing of ADAM9 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 #8754
Swiss-Prot Acc. #Q13443

Storage: ADAM9 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) N. M. Hooper and U. Lendeckel. The Adam Family Of Proteases. The Netherlands: Springer, 2005
- (2) Schlöndorff, J. and Blobel, C.P. (1999) *J Cell Sci* 112 (Pt 21), 3603-17.
- (3) Franzke, C.W. et al. (2002) *EMBO J* 21, 5026-35.
- (4) Zigrino, P. et al. (2007) *J Biol Chem* 282, 30785-93.
- (5) Fritzsche, F.R. et al. (2008) *Eur Urol* 54, 1097-106.
- (6) Grützmann, R. et al. (2004) *Br J Cancer* 90, 1053-8.
- (7) Carl-McGrath, S. et al. (2005) *Int J Oncol* 26, 17-24.
- (8) Shintani, Y. et al. (2004) *Cancer Res* 64, 4190-6.
- (9) Mazzocca, A. et al. (2005) *Cancer Res* 65, 4728-38.