**✓** 10 µM in 300 µl (100 transfections)



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

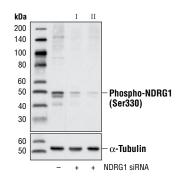
## Species Cross-Reactivity: H

Description: SignalSilence® NDRG1 siRNA I from Cell Signaling Technology (CST) allows the researcher to specifically inhibit NDRG1 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: NDRG1 (N-myc downstream-regulated gene 1), also termed Cap43, Drg1, RTP/rit42, and Proxy-1, is a member of the NDRG1 family comprised of four members (NDRG1-4) that function in growth, differentiation and cell survival (1-5). NDRG1 is ubiquitously expressed and highly responsive to a variety of stress signals including DNA damage (4), hypoxia (5) and elevated levels of nickel and calcium (2). Expression of NDRG1 is elevated in N-myc defective mice and is negatively regulated by N- and c-myc (1,6). During DNA damage NDRG1 is induced in a p53-dependent fashion and is necessary for p53-mediated apoptosis (4,7). NDRG1 may also play a role in cancer progression by promoting differentiation, inhibiting growth and modulating metastasis and angiogenesis (3,4,6,8,9). Nonsense mutation of the NDRG1 gene has been shown to cause hereditary motor and sensory neuropathy-Lom (HMSNL), which is supported by studies demonstrating the role of NDRG1 in maintaining myelin sheaths and axonal survival (10,11). NDRG1 is up-regulated during mast cell maturation and its deletion leads to attenuated allergic responses (12). Both NDGR1 and NDGR2 are substrates for SGK1 although the precise physiological role of this phosphorylation is not known (13). NDRG1 is phosphorylated by SGK1 at Thr328, Ser330, Thr346, Thr356, and Thr366. Phosphorylation by SGK1 primes NDRG1 for phosphorylation by GSK3.

Directions for Use: CST recommends transfection with 100 nM NDRG1 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.

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.



Western blot analysis of extracts from HeLa cells treated with Calyculin A #9902 (100nM for 20 minutes) and transfected with 100 nM SignalSilence® Control siRNA (Unconjugated) #6568 (-), SignalSilence® NDRG1 siRNA I (+) or SignalSilence® NDRG1 siRNA II #6257 (+), using Phospho-NDRG1 (Ser330) Antibody #3506 (upper) or  $\alpha$ -Tubulin (11H10) Rabbit mAb #2125 (lower). The Phospho-NDRG1 (Ser330) Antibody confirms silencing of NDRG1 expression, while the  $\alpha$ -Tubulin (11H10) Rabbit mAb is used a loading control.

Entrez-Gene ID #10397 Swiss-Prot Acc. #Q92597

Storage: NDRG1 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) Shimono, A. et al. (1999) Mech Dev 83, 39-52.
- (2) Zhou, D. et al. (1998) Cancer Res 58, 2182-9.
- (3) van Belzen, N. et al. (1997) Lab Invest 77, 85-92.
- (4) Kurdistani, S.K. et al. (1998) Cancer Res 58, 4439-44.
- (5) Park, H. et al. (2000) Biochem Biophys Res Commun 276,
- (6) Li, J. and Kretzner, L. (2003) Mol Cell Biochem 250, 91-105.
- (7) Stein, S. et al. (2004) J Biol Chem 279, 48930-40.
- (8) Maruyama, Y. et al. (2006) Cancer Res 66, 6233-42.
- (9) Nishio, S. et al. (2008) Cancer Lett 264, 36-43.
- (10) Kalaydjieva, L. et al. (2000) Am J Hum Genet 67, 47-58.
- (11) Okuda, T. et al. (2004) Mol Cell Biol 24, 3949-56.
- (12) Taketomi, Y. et al. (2007) J Immunol 178, 7042-53.
- (13) Murray, J.T. et al. (2004) Biochem J 384, 477-88.