

SignalSilence® p44 MAPK (Erk1) siRNA I



Cell Signaling
TECHNOLOGY®

- 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, (M, R, MK)

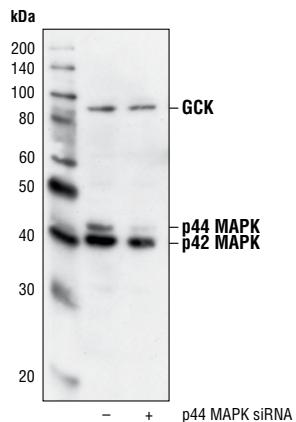
Description: SignalSilence® p44 MAPK (Erk1) siRNA I from Cell Signaling Technology (CST) allows the researcher to specifically inhibit p44 MAPK (Erk1) expression using RNA interference, a method whereby gene expression can be selectively silenced through the delivery of double stranded RNA molecules 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: Mitogen-activated protein kinases (MAPKs) are a widely conserved family of serine/threonine protein kinases involved in many cellular programs such as cell proliferation, differentiation, motility, and death. The p44/42 MAPK (Erk1/2) signaling pathway can be activated in response to a diverse range of extracellular stimuli including mitogens, growth factors, and cytokines (1-3) and is an important target in the diagnosis and treatment of cancer (4). Upon stimulation, a sequential three-part protein kinase cascade is initiated, consisting of a MAP kinase kinase kinase (MAPKKK or MAP3K), a MAP kinase kinase (MAPKK or MAP2K), and a MAP kinase (MAPK). Multiple p44/42 MAPKs have been identified, including members of the Raf family as well as Mos and Tpl2/Cot. MEK1 and MEK2 are the primary MAPKKs in this pathway (5,6). MEK1 and MEK2 activate p44 and p42 through phosphorylation of activation loop residues Thr202/Tyr204 and Thr185/Tyr187, respectively. Several downstream targets of p44/42 have been identified, including p90RSK (7) and the transcription factor Elk-1 (8,9). p44/42 are negatively regulated by a family of dual-specificity (Thr/Tyr) MAPK phosphatases, known as DUSPs or MKPs (10), along with MEK inhibitors such as U0126 and PD98059.

Small interfering RNA (siRNA) has been used to specifically silence p44 MAPK (Erk1) expression in HEK293 cells (7).

Directions for Use: CST recommends transfection with 100 nM p44 MAPK 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, transfected with 100 nM SignalSilence® Control siRNA (Fluorescein Conjugate) #6201 (-) or SignalSilence® p44 MAPK (Erk1) siRNA I (+), using p44/42 MAPK (Erk1/2) Antibody #9102 and GCK Antibody #3782. The Erk1/2 antibody confirms silencing of Erk1 expression and GCK antibody is used to control for loading and specificity of p44 MAPK (Erk1) siRNA.

Specificity/ Sensitivity: p44 MAPK (Erk1) siRNA I will inhibit human, mouse, rat and monkey p44 MAPK (Erk1) expression.

Entrez-Gene ID # 5595
Swiss-Prot Acc. # P27361

Storage: p44 MAPK (Erk1) 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) Roux, P.P. and Blenis, J. (2004) *Microbiol Mol Biol Rev* 68, 320-44.
- (2) Baccarini, M. (2005) *FEBS Lett* 579, 3271-7.
- (3) Meloche, S. and Pouysségur, J. (2007) *Oncogene* 26, 3227-39.
- (4) Roberts, P.J. and Der, C.J. (2007) *Oncogene* 26, 3291-310.
- (5) Rubinfeld, H. and Seger, R. (2005) *Mol Biotechnol* 31, 151-74.
- (6) Murphy, L.O. and Blenis, J. (2006) *Trends Biochem Sci* 31, 268-75.
- (7) Dalby, K.N. et al. (1998) *J Biol Chem* 273, 1496-505.
- (8) Marais, R. et al. (1993) *Cell* 73, 381-93.
- (9) Kortenjann, M. et al. (1994) *Mol Cell Biol* 14, 4815-24.
- (10) Owens, D.M. and Keyse, S.M. (2007) *Oncogene* 26, 3203-13.
- (11) Castro-Obregon, S. et al. (2004) *J. Biol. Chem.* 279, 17543-53.