

SignalSilence® PAK1 siRNA I



Orders ■ 877-616-CELL (2355)
orders@cellsignal.com
Support ■ 877-678-TECH (8324)
info@cellsignal.com
Web ■ www.cellsignal.com

rev. 02/10/16

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

Species Cross-Reactivity: H, (R)

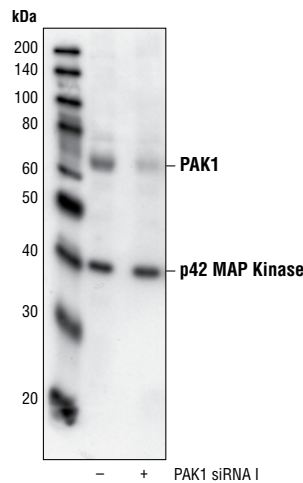
Description: SignalSilence® PAK1 siRNA I from Cell Signaling Technology (CST) allows the researcher to specifically inhibit PAK1 expression using RNA interference, a method whereby gene expression can be selectively silenced through the delivery of double stranded RNA molecules into the cell. All SignalSilence® siRNA products from CST are rigorously tested in-house and have been shown to reduce target protein expression by western analysis.

Background: The p21-activated kinase (PAK) family of serine/threonine kinases is engaged in multiple cellular processes, including cytoskeletal reorganization, MAPK signaling, apoptotic signaling, control of phagocyte NADPH oxidase and growth factor-induced neurite outgrowth (1,2). Several mechanisms that induce PAK activity have been reported. Binding of Rac/cdc42 to the CRIB (or PBD) domain near the amino terminus of PAK causes autophosphorylation and conformational changes in PAK (1). Phosphorylation of PAK1 at Thr423 by PDK induces activation of PAK1 (3). Several autophosphorylation sites have been identified, including serines 199 and 204 of PAK1 and serines 192 and 197 of PAK2 (4,5). Because the autophosphorylation sites are located in the amino-terminal inhibitory domain, it has been hypothesized that modification in this region prevents the kinase from reverting to an inactive conformation (6). Research indicates that phosphorylation of Ser144 of PAK1 or Ser139 of PAK3 (located in the kinase inhibitory domain) affects kinase activity (7). Phosphorylation of Ser21 of PAK1 or Ser20 of PAK2 regulates binding with the adaptor protein Nck (8). More recently identified family members including PAK4, PAK5 and PAK6 have lower sequence similarity with PAK1-3 in the amino-terminal regulatory region (9). Phosphorylation of Ser474 of PAK4, a site analogous to Thr423 of PAK1, may play a pivotal role in regulating the activity and function of PAK4 (10).

Small interfering RNA (siRNA) has been used to specifically silence PAK1 expression in MCF-7 cells (11).

Directions for Use: CST recommends transfection with 100nM PAK1 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® PAK1 siRNA I (+), using PAK1 Antibody #2602 and p42 MAP Kinase (Erk2) Antibody #9108. The PAK1 antibody confirms silencing of PAK1 expression, while the p42 MAP kinase antibody is used to control for loading and specificity of PAK1 siRNA.

Specificity/ Sensitivity: PAK1 siRNA I will inhibit human and rat PAK1 expression.

Entrez-Gene ID #5058
Swiss-Prot Acc. #Q13153

Storage: PAK1 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) Knaus, U.G. and Bokoch, G.M. (1998) *Int. J. Biochem. Cell Biol.* 30, 857-862.
- (2) Daniels, R.H. et al. (1998) *EMBO J.* 17, 754-764.
- (3) King, C.C. et al. (2000) *J. Biol. Chem.* 275, 41201-41209.
- (4) Manser, E. et al. (1997) *Mol. Cell. Biol.* 17, 1129-1143.
- (5) Gatti, A. et al. (1999) *J. Biol. Chem.* 274, 8022-8028.
- (6) Lei, M. et al. (2000) *Cell* 102, 387-397.
- (7) Chong, C. et al. (2001) *J. Biol. Chem.* 276, 17347-17353.
- (8) Zhao, Z. et al. (2000) *Mol. Cell. Biol.* 20, 3906-3917.
- (9) Abo, A. et al. (1998) *EMBO J.* 17, 6527-6540.
- (10) Qu, J. et al. (2001) *Mol. Cell. Biol.* 21, 3523-3533.
- (11) Wang, R. et al. (2002) *EMBO J.* 21, 5437-5447.