

# SignalSilence® Chk1 siRNA I

✓ 10 µM in 300 µl  
(100 Transfections)

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This product is intended for research purposes only. This product is not intended to be used for therapeutic or diagnostic purposes in humans or animals.

## Species Cross-Reactivity: H, Mk

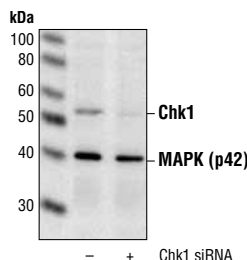
**Description:** SignalSilence® Chk1 siRNA from Cell Signaling Technology allows the researcher to specifically inhibit Chk1 expression using RNA interference, a method in which gene expression can be selectively silenced through the delivery of double stranded RNA molecules into the cell. All SignalSilence® siRNA products are rigorously tested in-house and have been shown to reduce protein expression in specified cell lines.

**Background:** Chk1 kinase acts downstream of ATM/ATR kinase to play an important role in DNA damage checkpoint control, embryonic development and tumor suppression (1). Activation of Chk1 involves phosphorylation of Ser317 and Ser345 and occurs in response to blocked DNA replication and certain forms of genotoxic stress (2). Phosphorylation at Ser 345 serves to localize Chk1 to the nucleus (3) following checkpoint activation while recently phosphorylation at Ser 317 along with site-specific phosphorylation of PTEN was shown to allow for reentry into the cell cycle following stalled DNA replication. (4). Chk1 exerts its checkpoint mechanism on the cell cycle in part by regulating the cdc25 family of phosphatases. Chk1 phosphorylation of cdc25A targets it for proteolysis and inhibits its activity through 14-3-3 binding. (5). Activated Chk1 can inactivate cdc25C via phosphorylation at Ser216, blocking the activation of cdc2 and transition into mitosis (6). Also, centrosomal Chk1 has been shown to phosphorylate cdc25B inhibiting its activation of CDK1-cyclin B1 and thus mitotic spindle formation and chromatin condensation (7). Furthermore, Chk1 plays a role in spindle checkpoint function through regulation of Aurora B and BubR1 (8). Chk1 has emerged as a drug target for cancer therapy as its inhibition leads to cell death in many cancer cell lines (9).

**Directions for Use:** CST recommends transfection with 50 nM Chk1 siRNA 48-72 hours prior to cell lysis.

**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.

**Specificity/ Sensitivity:** Chk1 siRNA I will inhibit human and monkey Chk1 expression.



Western blot analysis of extracts from HeLa cells transfected with non-targeted (-) or targeted (+) siRNA. Chk1 was detected using the Chk1 Antibody #2345, and p42 was detected using the p42 MAPK Antibody #9108. The Chk1 Antibody confirms silencing of Chk1 expression, and the p42 MAPK Antibody was used to control for loading and specificity of Chk1 siRNA.

Entrez-Gene ID #6198  
Swiss-Prot Acc. #P23443

**Storage:** Chk1 siRNA is supplied in RNase-free water. Aliquot and store at -20°C.

Please visit [www.cellsignal.com](http://www.cellsignal.com) for a complete listing of recommended companion products.

## Background References:

- (1) Liu, Q. et al. (2000) *Genes Dev* 14, 1448-59.
- (2) Zhao, H. and Piwnicka-Worms, H. (2001) *Mol Cell Biol* 21, 4129-39.
- (3) Jiang, K. et al. (2003) *J Biol Chem* 278, 25207-17.
- (4) Martin, S.A. and Ouchi, T. (2008) *Mol Cancer Ther* 7, 2509-16.
- (5) Chen, M.S. et al. (2003) *Mol Cell Biol* 23, 7488-97.
- (6) Zeng, Y. et al. (1998) *Nature* 395, 507-10.
- (7) Löffler, H. et al. (2006) *Cell Cycle* 5, 2543-7.
- (8) Zachos, G. et al. (2007) *Dev Cell* 12, 247-60.
- (9) Garber, K. (2005) *J Natl Cancer Inst* 97, 1026-8.
- (10) Chen, Z. et al. (2003) *Mol. Cancer Ther.* 2(6), 543-548.