

SignalSilence® PLK1 siRNA I



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

✓ 10 µM in 300 µl
(3 nmol)

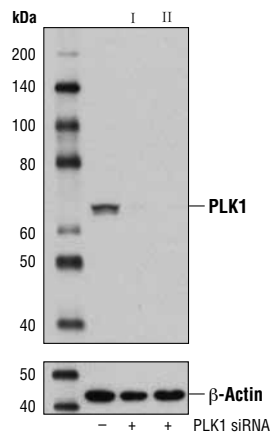
Species Cross-Reactivity: H, (Mk)

Description: SignalSilence® PLK1 siRNA I from Cell Signaling Technology (CST) allows the researcher to specifically inhibit PLK1 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: At least 4 distinct polo-like kinases exist in mammalian cells: PLK1, PLK2, PLK3, and PLK4/SAK (1). PLK1 apparently plays many roles during mitosis, particularly in regulating mitotic entry and exit. The mitosis promoting factor (MPF), cdc2/cyclin B1, is activated by dephosphorylation of cdc2 (Thr14/Tyr15) by cdc25C. PLK1 phosphorylates cdc25C at Ser198 and cyclin B1 at Ser133 causing translocation of these proteins from the cytoplasm to the nucleus (2-5). PLK1 phosphorylation of Myt1 at Ser426 and Thr495 has been proposed to inactivate Myt1, one of the kinases known to phosphorylate cdc2 at Thr14/Tyr15 (6). Polo-like kinases also phosphorylate the cohesin subunit SCC1, causing cohesin displacement from chromosome arms that allow for proper cohesin localization to centromeres (7). Mitotic exit requires activation of the anaphase promoting complex (APC) (8), a ubiquitin ligase responsible for removal of cohesin at centromeres, and degradation of securin, cyclin A, cyclin B1, Aurora A, and cdc20 (9). PLK1 phosphorylation of the APC subunits Apc1, cdc16, and cdc27 has been demonstrated *in vitro* and has been proposed as a mechanism by which mitotic exit is regulated (10,11).

Substitution of Thr210 with Asp has been reported to elevate PLK1 kinase activity and delay/arrest cells in mitosis, while a Ser137Asp substitution leads to S-phase arrest (12). In addition, while DNA damage has been found to inhibit PLK1 kinase activity, the Thr210Asp mutant is resistant to this inhibition (13). PLK1 has been reported to be phosphorylated *in vivo* at Ser137 and Thr210 in mitosis; DNA damage prevents phosphorylation at these sites (14).

Specificity/Sensitivity: SignalSilence® PLK1 siRNA I inhibits human and monkey PLK1 expression.



Western blot analysis of extracts from HeLa cells, transfected with 100 nM SignalSilence® Control siRNA (Unconjugated) #6568 (-), SignalSilence® PLK1 siRNA I (+), or SignalSilence® PLK1 siRNA II #12132 (+), using PLK1 (208G4) Rabbit mAb #4513 (upper) or β-Actin (D6A8) Rabbit mAb #8457 (lower). The PLK1 (208G4) Rabbit mAb confirms silencing of PLK1 expression, while the β-Actin (D6A8) Rabbit mAb is used as a loading control.

Directions for Use: CST recommends transfection with 100 nM SignalSilence® PLK1 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.

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.

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 #5347
Swiss-Prot Acc. #P53350

Storage: PLK1 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:

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- (12) Jang, Y.J. et al. (2002) *J Biol Chem* 277, 44115–44120.
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