Revision 1

Store at -20C

#5472

Phospho-PLK1 (Thr210) Antibody

Cell Signaling TECHNOLOGY®

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For Research Use Only. Not for Use in Diagnostic Procedures.

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W	H	Endogenous	62	Rabbit	#P53350	5347

Product Usage Information

Application	Dilution
Western Blotting	1:1000

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

Phospho-PLK1 (Thr210) Antibody detects endogenous levels PLK1 only when phosphorylated at threonine 210.

Species predicted to react based on 100% sequence homology

Monkey

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic phospho-peptide corresponding to residues surrounding Thr210 of human PLK1. Antibodies are purified using protein A and peptide affinity chromatography.

Background

At least four 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).

Background References

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3. Toyoshima-Morimoto, F. et al. (2001) *Nature* 410, 215-20.
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5. Jackman, M. et al. (2003) *Nat Cell Biol* 5, 143-8.
6. Nakajima, H. et al. (2003) *J Biol Chem* 278, 25277-80.
7. Sumara, I. et al. (2002) *Mol Cell* 9, 515-25.
8. Hauf, S. et al. (2001) *Science* 293, 1320-3.
9. Peters, J.M. (1999) *Exp. Cell Res.* 248, 339-49.
10. Kraft, C. et al. (2003) *EMBO J* 22, 6598-609.
11. Kotani, S. et al. (1998) *Mol Cell* 1, 371-80.
12. Jang, Y.J. et al. (2002) *J Biol Chem* 277, 44115-20.
13. Smits, V.A. et al. (2000) *Nat Cell Biol* 2, 672-6.
14. Tsvetkov, L. and Stern, D.F. (2005) *Cell Cycle* 4, 166-71.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting

Cross-Reactivity Key

H: Human

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