

## Phospho-PLK1 (Thr210) Antibody (ELISA-Specific)



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or Research Use Only. Not for Use in Diagnostic Procedures.					
<b>Applications:</b> E-P	Reactivity: H	<b>MW (kDa):</b> N/A.	<b>Source/Isotype:</b> Rabbit	UniProt ID: #P53350	Entrez-Gene Id: 5347
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 (ELISA-Specific) is phospho-specific by ELISA, but detects multiple bands by Western blot.			
Species predicted to react based on 100% sequence homology		Mouse, Xenopus, Pig			
Source / Purification		Polyclonal antibodies are produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Thr210 of human PLK1. Antibodies are purified by protein A and peptide affinity chromatography.			
Description		This antibody is formulated in PBS (no BSA/no glycerol) and quality controlled for use in ELISA and other drug discovery applications. This is a sample antibody and intended for use by drug discovery scientists.			
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 <i>in vitro</i> and has been proposed as a mechanism by which mitotic exit is regulated (10,11).			
		in mitosis, while a Ser13 has been found to inhib PLK1 has been reported prevents phosphorylatio Substitution of Thr210 v in mitosis, while a Ser13 has been found to inhib PLK1 has been reported	87Asp substitution leads to bit PLK1 kinase activity, the I to be phosphorylated <i>in</i> on at these sites (14). with Asp has been reporte 87Asp substitution leads to bit PLK1 kinase activity, the	o S-phase arrest (12). Thr210Asp mutant in vivo at Ser137 and Tlendre delevate PLK1 kind of S-phase arrest (12). Thr210Asp mutant vivo at Ser137 and Tlendre delevate Thr210Asp mutant vivo at Ser137 and Tlendre delevate the	hase activity and delay/arrest cells. In addition, while DNA damage is resistant to this inhibition (13). hr210 in mitosis; DNA damage hase activity and delay/arrest cells. Additionally, while DNA damage is resistant to this inhibition (13). hr210 in mitosis, and DNA
Background References		<ol> <li>Nigg, E.A. (1998) Curr Opin Cell Biol 10, 776-83.</li> <li>Toyoshima-Morimoto, F. et al. (2002) EMBO Rep 3, 341-8.</li> <li>Toyoshima-Morimoto, F. et al. (2001) Nature 410, 215-20.</li> <li>Peter, M. et al. (2002) EMBO Rep 3, 551-6.</li> <li>Jackman, M. et al. (2003) Nat Cell Biol 5, 143-8.</li> <li>Nakajima, H. et al. (2003) J Biol Chem 278, 25277-80.</li> <li>Sumara, I. et al. (2002) Mol Cell 9, 515-25.</li> <li>Hauf, S. et al. (2001) Science 293, 1320-3.</li> <li>Peters, J.M. (1999) Exp. Cell Res. 248, 339-49.</li> <li>Kraft, C. et al. (2003) EMBO J 22, 6598-609.</li> <li>Kotani, S. et al. (1998) Mol Cell 1, 371-80.</li> <li>Jang, Y.J. et al. (2002) J Biol Chem 277, 44115-20.</li> <li>Smits, V.A. et al. (2000) Nat Cell Biol 2, 672-6.</li> <li>Tsvetkov, L. and Stern, D.F. (2005) Cell Cycle 4, 166-71.</li> </ol>			

15. Jang, Y.J. et al. (2002) J Biol Chem 277, 44115-20. 16. Smits, V.A. et al. (2000) Nat Cell Biol 2, 672-6. 17. 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).

**Applications Key** E-P: Peptide ELISA (DELFIA)

**Cross-Reactivity Key** H: Human

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