

Phospho-Chk1 (Ser345) (133D3) Rabbit



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Applications: /, W-S, IF-IC, FC-FP	Reactivity: H M R Mk	Sensitivity: Endogenous	MW (kDa): 56	Source/Isotype: Rabbit IgG	UniProt ID: #O14757	Entrez-Gene Id 1111
Product Usage Information		Application Western Blotting				Dilution 1:1000
		Simple Western™ Immunofluorescence	(Immunocytochem	istry)		1:10 - 1:50 1:50
		Flow Cytometry (Fixed/Permeabilized)			1:200	
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.				
		For a carrier free (BSA and azide free) version of this product see product #76784.				
Specificity/Sensitivity		Phospho-Chk1 (Ser345) (133D3) Rabbit mAb detects endogenous levels of Chk1 only when phosphorylated at serine 345.				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Ser345 of human Chk1.				
Background		Chk1 kinase acts downstream of ATM/ATR kinase and plays an important role in DNA damage checkpoint control, embryonic development, and tumor suppression (1). Activation of Chk1 involves phosphorylation at Ser317 and Ser345 by ATM/ATR, followed by autophosphorylation of Ser296. Activation occurs in response to blocked DNA replication and certain forms of genotoxic stress (2). While phosphorylation at Ser345 serves to localize Chk1 to the nucleus following checkpoint activation (3), phosphorylation at Ser317 along with site-specific phosphorylation of PTEN allows for re-entry 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). Centrosomal Chk1 has been shown to phosphorylate cdc25B and inhibit its activation of CDK1-cyclin B1, thereby abrogating mitotic spindle formation and chromatin condensation (7). Furthermore, Chk1 plays a role in spindle checkpoint function through regulation of aurora B and BubR1 (8). Research studies have implicated Chk1 as a drug target for cancer therapy as its inhibition leads to cell death in many cancer cell lines (9).				
Background Ref	erences	 Liu, Q. et al. (2000) Genes Dev 14, 1448-59. Zhao, H. and Piwnica-Worms, H. (2001) Mol Cell Biol 21, 4129-39. Jiang, K. et al. (2003) J Biol Chem 278, 25207-17. Martin, S.A. and Ouchi, T. (2008) Mol Cancer Ther 7, 2509-16. Chen, M.S. et al. (2003) Mol Cell Biol 23, 7488-97. Zeng, Y. et al. (1998) Nature 395, 507-10. Löffler, H. et al. (2006) Cell Cycle 5, 2543-7. Zachos, G. et al. (2007) Dev Cell 12, 247-60. Garber, K. (2005) J Natl Cancer Inst 97, 1026-8. 				

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 W-S: Simple Western™ IF-IC: Immunofluorescence (Immunocytochemistry) FC-FP:

Flow Cytometry (Fixed/Permeabilized)

Cross-Reactivity Key

H: Human M: Mouse R: Rat Mk: Monkey

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