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Phospho-Chk1 (Ser317) Antibody



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Entrez-Gene Id: Applications: Reactivity: Sensitivity: MW (kDa): Source: **UniProt ID:** WB H Mk Mi Endogenous 56 Rabbit #O14757 1111 **Product Usage** Application Dilution Information Western Blotting 1:1000 Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μg/ml BSA and 50% glycerol. Store at -**Storage** 20°C. Do not aliquot the antibody. Phospho-Chk1 (Ser317) Antibody detects endogenous levels of Chk1 only when phosphorylated at Specificity / Sensitivity Ser317. The antibody does not recognize Chk1 phosphorylated at other sites.

Polyclonal antibodies are produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Ser317 of human Chk1. Antibodies are purified by protein A and peptide affinity

chromatography.

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 References

Source / Purification

- 1. Liu, Q. et al. (2000) Genes Dev 14, 1448-59.
- 2. Zhao, H. and Piwnica-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.

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 nonfat dry

milk, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key WB: Western Blotting

Cross-Reactivity Key H: human M: mouse R: rat Hm: hamster Mk: monkey Vir: virus Mi: mink C: chicken Dm: D. melanogaster

X: Xenopus Z: zebrafish B: bovine Dg: dog Pg: pig Sc: S. cerevisiae Ce: C. elegans Hr: horse

GP: Guinea Pig Rab: rabbit All: all species expected

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