

PhosphoPlus® Chk1 (Ser317) Antibody Duet

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

UniProt ID:
#O14757

Entrez-Gene Id:
1111

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Chk1 (2G1D5) Mouse mAb	2360	100 µl	56 kDa	Mouse IgG1
Phospho-Chk1 (Ser317) (D12H3) XP® Rabbit mAb	12302	100 µl	56 kDa	Rabbit IgG

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

PhosphoPlus® Duets from Cell Signaling Technology (CST) provide a means to assess protein activation status. Each Duet contains an activation-state and total protein antibody to your target of interest. These antibodies have been selected from CST's product offering based upon superior performance in specified applications.

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 antibodies.*

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

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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.

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