Revision 1

Store at -20C

#8038

Phospho-CDC20 (Ser51) 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, IP	H M	Endogenous	51	Rabbit	#Q12834	991

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:50

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-CDC20 (Ser51) Antibody recognizes endogenous levels of CDC20 protein only when phosphorylated at Ser51.

Species predicted to react based on 100% sequence homology

Rat

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Ser51 of human CDC20 protein. Antibodies are purified by protein A and peptide affinity chromatography.

Background

The cell division cycle demands accuracy to avoid the accumulation of genetic damage. This process is controlled by molecular circuits called "checkpoints" that are common to all eukaryotic cells (1). Checkpoints monitor DNA integrity and cell growth prior to replication and division at the G1/S and G2/M transitions, respectively. The cdc2-cyclin B kinase is pivotal in regulating the G2/M transition (2,3). Cdc2 is phosphorylated at Thr14 and Tyr15 during G2-phase by the kinases Wee1 and Myt1, rendering it inactive. The tumor suppressor protein retinoblastoma (Rb) controls progression through the late G1 restriction point (R) and is a major regulator of the G1/S transition (4). During early and mid G1-phase, Rb binds to and represses the transcription factor E2F (5). The phosphorylation of Rb late in G1-phase by CDKs induces Rb to dissociate from E2F, permitting the transcription of S-phase-promoting genes. *In vitro*, Rb can be phosphorylated at multiple sites by cdc2, cdk2, and cdk4/6 (6-8). DNA damage triggers both the G2/M and the G1/S checkpoints. DNA damage activates the DNA-PK/ATM/ATR kinases, which phosphorylate Chk at Ser345 (9), Chk2 at Thr68 (10) and p53 (11). The Chk kinases inactivate cdc25 via phosphorylation at Ser216, blocking the activation of cdc2.
CDC20 binds to and activates the anaphase-promoting complex (APC) during mitosis and G1 phase of the cell cycle (12). Moreover, CDC20 is necessary for ubiquitin ligase activity of the APC/cyclosome (APC/C). In metaphase MAD2L1 inactivates the CDC20-APC/C complex, while in anaphase this inhibition is lost and CDC20-APC/C degrades its substrates (13). p53 and p21 suppress expression of CDC20 upon genotoxic stresses and ectopic introduction of p53. siRNA mediated knock-down of CDC20 in cancer cells leads to attenuated cell growth and induces G(2)/M arrest, suggesting that CDC20 is a possible therapeutic target of cancer (14). Organization of neuronal circuits requires presynaptic axonal differentiation and synapse formation. CDC20-APC regulates presynaptic differentiation in postmitotic neurons by triggering the required degradation of the transcription factor NeuroD2 (15).
Phosphorylation of CDC20 at Ser51 by CAMKIIβ disperses CDC20 from the centrosome. As a result, CDC20-APC activity is inhibited and transition from growth to retraction of dendrites in primary neurons is triggered (16).

Background References

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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 **IP:** Immunoprecipitation

Cross-Reactivity Key

H: Human **M:** Mouse

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Limited Uses