SANTA CRUZ BIOTECHNOLOGY, INC.

p-Cdk (Thr14/Tyr15)-R: sc-28435-R

SANTA CRUZ BIOTECHNOLOGY
The Power to Question

BACKGROUND

The phosphorylation and dephosphorylation of proteins on serine and threonine residues is an essential means of regulating a broad range of cellular functions in eukaryotes, including cell division, homeostasis and apoptosis. A group of proteins that are intimately involved in this process are the serine/threonine (Ser/Thr) protein kinases. In humans, the cyclin-dependent protein kinase complex is essential for cell-cycle control and is comprised of catalytic and regulatory proteins that function as Ser/Thr protein kinases. The catalytic cyclins, four of which are designated Cdc2 p34, Cdk2, Cdk3 and Cdk5, function at different stages of the cell cycle. Together, these proteins catalyze the ATP-dependent reactions that allow the cell to progress through the G_1/S-phase transition and, ultimately, through mitosis. The cyclin-dependent protein kinase complex is regulated by proteins such as p21 and p27 that associate with the complex and control its activity. Further regulation of the catalytic subunits is achieved via phosphorylation at residues such as Thr14 and Thr15, an event that either activates or deactivates the target protein.

REFERENCES

1. Braun, K., et al. 1998. Investigation of the cell cycle regulation of Cdk3-associated kinase activity and the role of Cdk3 in proliferation and transformation. Oncogene 17: 2259-2269.
2. Schang, L.M., et al. 2002. Explant-induced reactivation of herpes simplex virus occurs in neurons expressing nuclear Cdk2 and Cdk4. J. Virol. 76: 7724-7735.
3. Ren, S. and Rollins, B.J. 2004. Cyclin C/Cdk3 promotes Rb-dependent G_0 exit. Cell 117: 239-251.
4. Zhang, B., et al. 2007. The activation and inhibition of cyclin-dependent kinase-5 by phosphorylation. Biochemistry 46: 10841-10851.
5. Wissing, J., et al. 2007. Proteomics analysis of protein kinases by target class-selective prefractionation and tandem mass spectrometry. Mol. Cell. Proteomics 6: 537-547.

SOURCE

p-Cdk (Thr14/Tyr15)-R is a rabbit polyclonal antibody raised against a short amino acid sequence containing Thr 14 and Tyr 15 phosphorylated Cdk2 of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-28435 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

APPLICATIONS

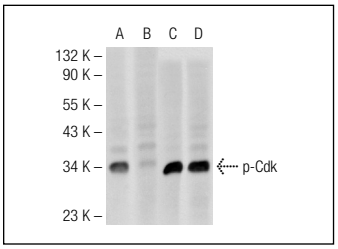
p-Cdk (Thr14/Tyr15)-R is recommended for detection of Thr14 and Tyr15 dually phosphorylated Cdk1, Cdk2, Cdk3 and Cdk5 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

p-Cdk (Thr14/Tyr15)-R is also recommended for detection of correspondingly dually phosphorylated Cdk1, Cdk2, Cdk3 and Cdk5 in additional species, including equine, canine, bovine, porcine and avian.

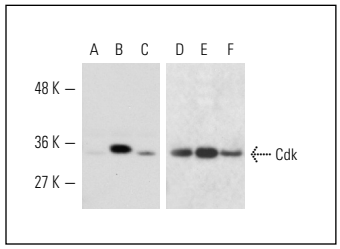
Molecular Weight of p-Cdk: 34 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200 or HeLa + serum starved cell lysate: sc-24693.

DATA



Western blot analysis of Cdk phosphorylation in untreated (**A**,**C**) and lambda protein phosphatase (sc-200312A) treated (**B**,**D**) HeLa whole cell lysates. Antibodies tested include p-Cdk (Thr14/Tyr15)-R: sc-28435-R (**A**,**B**) and Cdk2 (D-12): sc-6248 (**C**,**D**).



Western blot analysis of Cdk phosphorylation in HeLa (**A**,**D**), Nocodazole-treated HeLa (**B**,**E**) and serum starved HeLa (**C**,**F**) whole cell lysates. Antibodies tested include phospho-specific antibody, p-Cdk (Thr14/Tyr15)-R: sc-28435-R (**A**,**B**,**C**) and control antibody, Cdk2 (D-12): sc-6248 (**D**,**E**,**F**).

SELECT PRODUCT CITATIONS

1. Stolfi, C., et al. 2008. Mesalazine negatively regulates Cdc25A protein expression and promotes accumulation of colon cancer cells in S phase. Carcinogenesis 29: 1258-1266.
2. Stolfi, C., et al. 2009. Inhibition of colon carcinogenesis by 2-methoxy-5-amino-N-hydroxybenzamide, a novel derivative of mesalamine. Gastroenterology 138: 221-230.
3. Koledova, Z., et al. 2010. DNA damage-induced degradation of Cdc25A does not lead to inhibition of Cdk2 activity in mouse embryonic stem cells. Stem Cells 28: 450-461.
4. Hui, S., et al. 2011. Peptide-mediated disruption of calmodulin-cyclin E interactions inhibits proliferation of vascular smooth muscle cells and neointima formation. Circ. Res. 108: 1053-1062.
5. Shi, Z., et al. 2015. Cables1 complex couples survival signaling to the cell death machinery. Cancer Res. 75: 147-158.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.