SANTA CRUZ BIOTECHNOLOGY, INC.

p-Chk1 (Ser 345): sc-17922

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Cell cycle events are regulated by the sequential activation and deactivation of cyclin dependent kinases (Cdks) and by proteolysis of cyclins. Chk1 and Chk2 are involved in these processes as regulators of Cdks. Chk1 and Chk2 both function as essential components in the G_2 DNA damage checkpoint by phosphorylating Cdc25C in response to DNA damage. Phosphorylation inhibits Cdc25C activity, thereby blocking mitosis. Cdc25A, Cdc25B and Cdc25C protein tyrosine phosphatases function as mitotic activators by dephosphorylating Cdc2 p34 on regulatory tyrosine residues. It has also been shown that Chk1 can phosphorylate Wee 1 *in vitro,* providing evidence that the hyperphosphorylated form of Wee 1, seen in cells delayed by Chk1 overexpression, is due to phosphorylation by Chk1. Chk1 is phosphorylated on Serine 345 (S345) in response to UV, IR and hydroxyurea (HU). Chk1 plays an essential role in the mammalian DNA damage checkpoint, embryonic development and tumor suppression.

REFERENCES

1. Gautier, J., et al. 1991. Cdc25 is a specific tyrosine phosphatase that directly activates $p34^{Cdc2}$. Cell 67: 197-211.
2. Barinaga, M. 1995. A new twist to the cell cycle. Science 269: 631-632.
3. Sanchez, Y., et al. 1997. Conservation of the Chk1 checkpoint pathway in mammals: linkage of DNA damage to Cdk regulation through Cdc25. Science 277: 1497-1501.
4. O'Connell, M.J., et al. 1997. Chk1 is a Wee1 kinase in the G_2 DNA damage checkpoint inhibiting Cdc2 by Y15 phosphorylation. EMBO J. 16: 545-554.
5. Peng, C.Y., et al. 1997. Mitotic and G_2 checkpoint control: regulation of 14-3-3 protein binding by phosphorylation of Cdc25C on Serine 216. Science 277: 1501-1505.

CHROMOSOMAL LOCATION

Genetic locus: CHEK1 (human) mapping to 11q24.2; Chek1 (mouse) mapping to 9 A4.

SOURCE

p-Chk1 (Ser 345) is available as either goat (sc-17922) or rabbit (sc-17922-R) polyclonal affinity purified antibody raised against a short amino acid sequence containing Ser 345 phosphorylated Chk1 of human origin.

PRODUCT

Each vial contains either 100 µg (sc-17922) or 200 µg (sc-17922-R) IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-17922 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.

APPLICATIONS

p-Chk1 (Ser 345) is recommended for detection of Ser 345 phosphorylated Chk1 of mouse, rat, human and *Xenopus laevis* origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), 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-Chk1 (Ser 345) is also recommended for detection of correspondingly phosphorylated Chk1 in additional species, including equine, canine, bovine, porcine and avian.

Suitable for use as control antibody for Chk1 siRNA (h): sc-29269, Chk1 siRNA (m): sc-29270, Chk1 shRNA Plasmid (h): sc-29269-SH, Chk1 shRNA Plasmid (m): sc-29270-SH, Chk1 shRNA (h) Lentiviral Particles: sc-29269-V and Chk1 shRNA (m) Lentiviral Particles: sc-29270-V.

Molecular Weight of p-Chk1: 56 kDa.

SELECT PRODUCT CITATIONS

1. Singh, S.V., et al. 2004. Sulforaphane-induced G_2/M phase cell cycle arrest involves checkpoint kinase 2-mediated phosphorylation of cell division cycle 25C. J. Biol. Chem. 279: 25813-25822.
2. Wu, Q., et al. 2008. Human MLH1 protein participates in genomic damage checkpoint signaling in response to DNA interstrand crosslinks, while MSH2 functions in DNA repair. PLoS Genet. 4: e1000189.
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4. Mazumder, D., et al. 2010. Inactivation of CHEK1 and EI24 are associated with the development of invasive cervical carcinoma: clinical and prognostic implications. Int. J. Cancer 129: 1859-1871.
5. Wiegant, W.W., et al. 2010. A novel radiosensitive SCID patient with a pronounced G_2/M sensitivity. DNA Repair 9: 365-373.
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7. Karimi-Busheri, F., et al. 2010. Senescence evasion by MCF-7 human breast tumor-initiating cells. Breast Cancer Res. 12: R31.
8. Zhang, L., et al. 2010. Proteolysis of Rad17 by Cdh1/APC regulates checkpoint termination and recovery from genotoxic stress. EMBO J. 29: 1726-1737.
9. Zhou, Z., et al. 2013. Regulation of Rad17 protein turnover unveils an impact of Rad17-APC cascade in breast carcinogenesis and treatment. J. Biol. Chem. 288: 18134-18145.
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RESEARCH USE

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