

# p-Cdc25C (Ser 216): sc-12354

## BACKGROUND

The Cdc2/cyclin B enzyme, involved in regulating mitosis in eukaryotic cells, is subject to multiple levels of control. Among these, the regulation of the catalytic subunit by tyrosine phosphorylation is the best understood. Tyrosine phosphorylation inhibits the Cdc2/cyclin B complex, while tyrosine dephosphorylation, which occurs at the onset of mitosis, directly activates the pre-MPH complex. The tyrosine phosphatase Cdc25 serves as a rate-limiting mitotic activator by regulating Cdc2 tyrosine phosphatase. In the absence of Cdc25, Cdc2 accumulates in a tyrosine phosphorylated state. In addition, Cdc25 proteins from a variety of species have been shown to share a low degree of sequence similarity with other tyrosine phosphatases. Activation of the Cdc25C is proposed to occur through an autocatalytic feedback loop mechanism involving nuclear Cdc2. The nuclear accumulation of Cdc25C is negatively regulated by phosphorylation at Ser 216. This phosphorylation event enables binding to 14-3-3 proteins, which then shuttle the phosphorylated Cdc25 out of the nucleus after DNA damage.

## CHROMOSOMAL LOCATION

Genetic locus: CDC25C (human) mapping to 5q31.2.

## SOURCE

p-Cdc25C (Ser 216) is available as either goat (sc-12354) or rabbit (sc-12354-R) polyclonal affinity purified antibody raised against a short amino acid sequence containing phosphorylated Ser 216 of Cdc25C 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-12354 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

## APPLICATIONS

p-Cdc25C (Ser 216) is recommended for detection of Ser 216 phosphorylated Cdc25C of 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).

Suitable for use as control antibody for Cdc25C siRNA (h): sc-35038, Cdc25C shRNA Plasmid (h): sc-35038-SH and Cdc25C shRNA (h) Lentiviral Particles: sc-35038-V.

Molecular Weight of p-Cdc25C: 55 kDa.

Positive Controls: K-562 whole cell lysate: sc-2203, PC-3 cell lysate: sc-2220 or HeLa whole cell lysate: sc-2200.

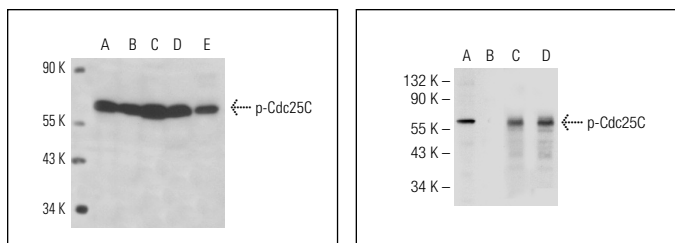
## STORAGE

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.

## DATA



p-Cdc25C (Ser 216)-R: sc-12354-R. Western blot analysis of Cdc25C phosphorylation in K-562 (A), PC-3 (B), Raji (C), untreated HeLa (D) and nocodazole-treated HeLa (E) whole cell lysates.

Western blot analysis of Cdc25C phosphorylation in untreated (A, C), and lambda protein phosphatase (sc-200312A) treated (B, D) HeLa whole cell lysates. Antibodies tested include p-Cdc25C (Ser 216)-R: sc-12354-R (A, C) and Cdc25C (C-20): sc-327 (B, D).

## SELECT PRODUCT CITATIONS

- Hapke, G., et al. 2002. Phosphorylation of Chk1 at Serine 345 affected by topoisomerase I poison SN-38. *Int. J. Oncol.* 21: 1059-1066.
- Mansour, A., et al. 2004. Genistein induces G<sub>2</sub> arrest in malignant B cells by decreasing IL-10 secretion. *Cell Cycle* 3: 1597-1605.
- Singh, S.V., et al. 2004. Sulforaphane-induced G<sub>2</sub>/M phase cell cycle arrest involves checkpoint kinase 2-mediated phosphorylation of cell division cycle 25C. *J. Biol. Chem.* 279: 25813-25822.
- Herman-Antosiewicz, A., et al. 2005. Checkpoint kinase 1 regulates diallyl trisulfide-induced mitotic arrest in human prostate cancer cells. *J. Biol. Chem.* 280: 28519-28528.
- Xiao, D., et al. 2005. Diallyl trisulfide-induced G<sub>2</sub>-M phase cell cycle arrest in human prostate cancer cells is caused by reactive oxygen species-dependent destruction and hyperphosphorylation of Cdc 25 C. *Oncogene* 24: 6256-6268.
- Tsou, T.C., et al. 2006. ATM/ATR-related checkpoint signals mediate arsenite-induced G<sub>2</sub>/M arrest in primary aortic endothelial cells. *Arch. Toxicol.* 80: 804-810.
- Ismail, I.A., et al. 2007. Genistein-induced neuronal apoptosis and G<sub>2</sub>/M cell cycle arrest is associated with MDC1 up-regulation and Plk1 down-regulation. *Eur. J. Pharmacol.* 575: 12-20.
- Zheng, M., et al. 2008. Growth inhibition and radiosensitization of glioblastoma and lung cancer cells by small interfering RNA silencing of tumor necrosis factor receptor-associated factor 2. *Cancer Res.* 68: 7570-7578.
- Wang, J., et al. 2009. Identification of XAF1 as a novel cell cycle regulator through modulating G<sub>2</sub>/M checkpoint and interaction with checkpoint kinase 1 in gastrointestinal cancer. *Carcinogenesis* 30: 1507-1516.
- Carrassa, L., et al. 2010. Role of Chk1 in the differentiation program of hematopoietic stem cells. *Cell. Mol. Life Sci.* 67: 1713-1722.