SANTA CRUZ BIOTECHNOLOGY, INC.

p16 INK4A (F-9): sc-55600

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The progression of cells through the cell cycle is regulated by a family of protein kinases known as cyclin-dependent kinases (Cdks). The sequential activation of individual members of this family and their consequent phosphorylation of critical substrates promotes orderly progression through the cell cycle. The cyclins function as differentially expressed positive regulators of Cdks. Negative regulators of the cycle include the p53-inducible protein p21 Waf1/Cip1 (also designated p21, WAF1 or Cip1), Kip1 p27 and p16 INK4A. The complexes formed by Cdk4 and the D-type cyclins have been strongly implicated in the control of cell proliferation during the G_1 phase. It has been shown that p16 INK4A binds to Cdk4 and inhibits the catalytic activity of the Cdk4/cyclin D complex. Moreover, the gene encoding p16 INK4A exhibits a high frequency of homozygous deletions and point mutations in established human tumor cell lines.

REFERENCES

1. Sherr, C.J. 1993. Mammalian G_1 cyclins. Cell 73: 1059-1065.
2. Harper, J.W., et al. 1993. The p21 Cdk-interacting protein Cip1 is a potent inhibitor of G_1 cyclin-dependent kinases. Cell 75: 805-816.
3. El-Deiry, W.S., et al. 1993. WAF1, a potential mediator of p53 tumor suppression. Cell 75: 817-825.
4. Hunter, T. 1993. Braking the cycle. Cell 75: 839-841.
5. Xiong, Y., et al. 1993. p21 is a universal inhibitor of cyclin kinases. Nature 366: 701-704.
6. Serrano, M., et al. 1993. A new regulatory motif in cell cycle control causing specific inhibition of cyclin D/Cdk4. Nature 366: 704-707.
7. Polyak, K., et al. 1994. p27 Kip1, a cyclin-Cdk inhibitor, links transforming growth factor β and contact inhibition to cell cycle arrest. Genes Dev. 8: 9-22.

CHROMOSOMAL LOCATION

Genetic locus: Cdkn2a (mouse) mapping to 4 C4.

SOURCE

p16 INK4A (F-9) is a mouse monoclonal antibody raised against amino acids 1-168 representing full length p16 INK4A of mouse origin.

PRODUCT

Each vial contains 200 µg IgG_{2a} in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

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

PROTOCOLS

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

APPLICATIONS

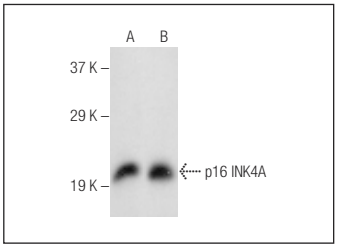
p16 INK4A (F-9) is recommended for detection of p16 INK4A of mouse origin by Western Blotting (starting dilution 1:100, dilution range 1:100), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for p16 INK4A siRNA (m): sc-36144, p16 INK4A shRNA Plasmid (m): sc-36144-SH and p16 INK4A shRNA (m) Lentiviral Particles: sc-36144-V.

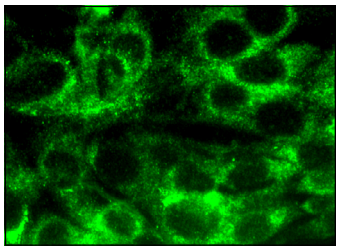
Molecular Weight of p16 INK4A: 16 kDa.

Positive Controls: 3T3-L1 cell lysate: sc-2243, MM-142 cell lysate: sc-2246 or WEHI-3 cell lysate: sc-3815.

DATA



p16 INK4A (F-9): sc-55600. Western blot analysis of p16 INK4A expression in 3T3-L1 (**A**) and mouse fibroblast (**B**) whole cell lysates.



p16 INK4A (F-9): sc-55600. Immunofluorescence staining of methanol-fixed NIH/3T3 cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Wan, X., et al. 2014. Mll2 controls cardiac lineage differentiation of mouse embryonic stem cells by promoting H3K4me3 deposition at cardiac-specific genes. Stem Cell Rev. Rep. 10: 643-652.
2. Ding, X., et al. 2015. Mixed lineage leukemia 5 (MLL5) protein stability is cooperatively regulated by O-GlcNac transferase (OGT) and ubiquitin specific protease 7 (USP7). PLoS ONE 10: e0145023.
3. Quijada, P., et al. 2015. Nuclear calcium/calmodulin-dependent protein kinase II signaling enhances cardiac progenitor cell survival and cardiac lineage commitment. J. Biol. Chem. 290: 25411-25426.
4. Kipkeew, F., et al. 2016. CCN1 (CYR61) and CCN3 (NOV) signaling drives human trophoblast cells into senescence and stimulates migration properties. Cell Adh. Migr. 10: 163-178.
5. Mitry, M.A., et al. 2020. Accelerated cardiomyocyte senescence contributes to late-onset doxorubicin-induced cardiotoxicity. Am. J. Physiol., Cell Physiol. 318: C380-C391.

RESEARCH USE

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