SANTA CRUZ BIOTECHNOLOGY, INC.

p16 INK4A (F-4): sc-74401

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.

CHROMOSOMAL LOCATION

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

SOURCE

p16 INK4A (F-4) 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_1 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

p16 INK4A (F-4) is recommended for detection of p16 INK4A of mouse origin by Western Blotting (starting dilution 1:100, 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 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 mouse LacZ whole cell lysate: sc-364371.

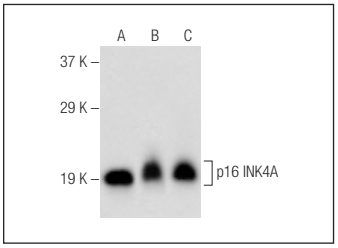
RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use m-IgGκ BP-FITC: sc-516140 or m-IgGκ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz® Mounting Medium: sc-24941 or UltraCruz® Hard-set Mounting Medium: sc-359850.

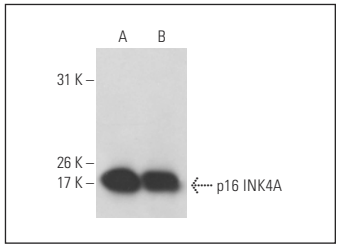
STORAGE

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

DATA



p16 INK4A (F-4): sc-74401. Western blot analysis of p16 INK4A expression in 3T3-L1 (**A**), MM-142 (**B**) and mouse fibroblast (**C**) whole cell lysates.



p16 INK4A (F-4): sc-74401. Western blot analysis of p16 INK4A expression in 3T3-L1 (**A**) and mouse LacZ (**B**) whole cell lysates.

SELECT PRODUCT CITATIONS

1. Rodriguez, R., et al. 2009. Loss of p53 induces tumorigenesis in p21-deficient mesenchymal stem cells. Neoplasia 11: 397-407.
2. Tang, X.H., et al. 2009. A DNA methyltransferase inhibitor and all-*trans* retinoic acid reduce oral cavity carcinogenesis induced by the carcinogen 4-nitroquinoline 1-oxide. Cancer Prev. Res. 2: 1100-1110.
3. Gao, J., et al. 2010. Fullerene derivatives induce premature senescence: a new toxicity paradigm or novel biomedical applications. Toxicol. Appl. Pharmacol. 244: 130-143.
4. Sideridou, M., et al. 2011. Cdc6 expression represses E-cadherin transcription and activates adjacent replication origins. J. Cell Biol. 195: 1123-1140.
5. Lee, M.E., et al. 2012. Cyclooxygenase-2 inhibitors modulate skin aging in a catalytic activity-independent manner. Exp. Mol. Med. 44: 536-544.
6. Fry, E.A., et al. 2013. Dmp1α inhibits HER2/neu-induced mammary tumorigenesis. PLoS ONE 8: e77870.
7. Zhu, S., et al. 2013. Cooperation between Dmp1 loss and cyclin D1 overexpression in breast cancer. Am. J. Pathol. 183: 1339-1350.
8. Hilgendorf, K.I., et al. 2013. The retinoblastoma protein induces apoptosis directly at the mitochondria. Genes Dev. 27: 1003-1015.
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11. Al-Qasem, A.J., et al. 2022. Co-targeting CDK2 and CDK4/6 overcomes resistance to aromatase and CDK4/6 inhibitors in ER+ breast cancer. NPJ Precis. Oncol. 6: 68.

RESEARCH USE

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