SANTA CRUZ BIOTECHNOLOGY, INC.

p16 INK4A (JC8): sc-56330

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 (human) mapping to 9p21.3.

SOURCE

p16 INK4A (JC8) is a mouse monoclonal antibody raised against full length recombinant p16 INK4A of human origin.

PRODUCT

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

p16 INK4A (JC8) is available conjugated to agarose (sc-56330 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-56330 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-56330 PE), fluorescein (sc-56330 FITC), Alexa Fluor® 488 (sc-56330 AF488), Alexa Fluor® 546 (sc-56330 AF546), Alexa Fluor® 594 (sc-56330 AF594) or Alexa Fluor® 647 (sc-56330 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-56330 AF680) or Alexa Fluor® 790 (sc-56330 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

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APPLICATIONS

p16 INK4A (JC8) is recommended for detection of p16 INK4A of human origin by Western Blotting (starting dilution to be determined by researcher, dilution range 1:10-1:200), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution to be determined by researcher, dilution range 1:10-1:200), immunohistochemistry (including paraffin-embedded sections) (starting dilution to be determined by researcher, dilution range 1:10-1:200) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for p16 INK4A siRNA (h): sc-36143, p16 INK4A shRNA Plasmid (h): sc-36143-SH and p16 INK4A shRNA (h) Lentiviral Particles: sc-36143-V.

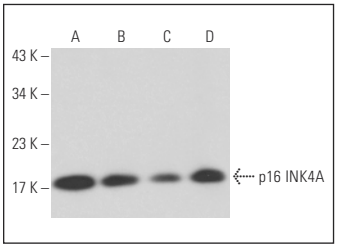
Molecular Weight of p16 INK4A: 16 kDa.

Positive Controls: Saos-2 cell lysate: sc-2235, HeLa whole cell lysate: sc-2200 or SHP-77 whole cell lysate: sc-364258.

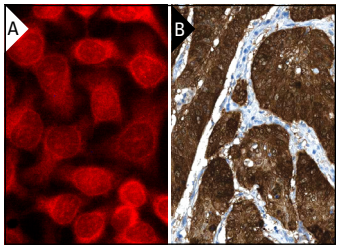
STORAGE

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

DATA



p16 INK4A (JC8): sc-56330. Western blot analysis of p16 INK4A expression in HeLa (**A**), Saos-2 (**B**), SHP-77 (**C**) and ME-180 (**D**) whole cell lysates.



p16 INK4A (JC8): sc-56330. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic and nuclear localization (**A**). Immunoperoxidase staining of formalin fixed, paraffin-embedded human cervical cancer tissue showing nuclear and cytoplasmic staining of tumor cells. Kindly provided by The Swedish Human Protein Atlas (HPA) program (**B**).

SELECT PRODUCT CITATIONS

1. Romanenko, A., et al. 2002. P16INK4A and p15INK4B gene alteration associated with oxidative stress in renal cell carcinomas after the chernobyl accident (pilot study). Diagn. Mol. Pathol. 11: 163-169.
2. Sznurkowski, J.J., et al. 2017. Local immune response depends on p16 INK4A status of primary tumor in vulvar squamous cell carcinoma. Oncotarget 8: 46204-46210.
3. Huang, W.B., et al. 2018. Human papillomavirus and World Health Organization type III nasopharyngeal carcinoma: multicenter study from an endemic area in Southern China. Cancer 124: 530-536.
4. De Cecco, M., et al. 2019. L1 drives IFN in senescent cells and promotes age-associated inflammation. Nature 566: 73-78.
5. Xu, J., et al. 2020. Cullin-7 (CUL7) is overexpressed in glioma cells and promotes tumorigenesis via NFκB activation. J. Exp. Clin. Cancer Res. 39: 59.
6. Liu, P., et al. 2021. m^6A-independent genome-wide METTL3 and METTL14 redistribution drives the senescence-associated secretory phenotype. Nat. Cell Biol. 23: 355-365.
7. Riess, C., et al. 2022. Implementation of a combined CDK inhibition and arginine-deprivation approach to target arginine-auxotrophic glioblastoma multiforme cells. Cell Death Dis. 13: 555.
8. Zhang, Z., et al. 2023. Induction of senescence by loss of Gata4 in cardiac fibroblasts. Cells 12: 1652.
9. Ma, X., et al. 2024. TRPM7 controls skin keratinocyte senescence by targeting intracellular calcium signaling. FEBS J. 291: 4680-4695.

RESEARCH USE

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