SANTA CRUZ BIOTECHNOLOGY, INC.

p16 (C-20): sc-468

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 (also designated WAF1 or CIP1), Kip1 p27 and p16. 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 binds to Cdk4 and inhibits the catalytic activity of the Cdk4/cyclin D complex. Moreover, the gene encoding p16 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.

CHROMOSOMAL LOCATION

Genetic locus: CDKN2A (human) mapping to 9p21.3.

SOURCE

p16 (C-20) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of p16 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-468 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

p16 (C-20) is recommended for detection of p16 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 p16 siRNA (h): sc-36143, p16 shRNA Plasmid (h): sc-36143-SH and p16 shRNA (h) Lentiviral Particles: sc-36143-V

Molecular Weight of p16: 16 kDa.

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

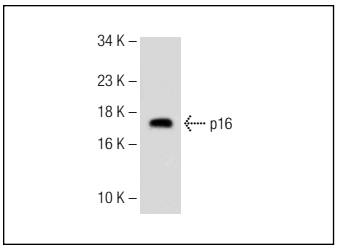
RESEARCH USE

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

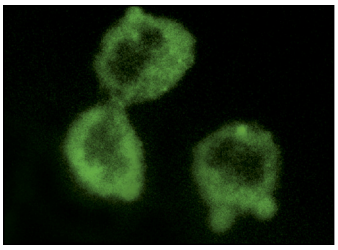
STORAGE

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

DATA



p16 (C-20)-G: sc-468-G. Western blot analysis of p16 expression in SHP-77 whole cell lysate.



p16 (C-20): sc-468. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Bhunia, A.K., et al. 2002. PKD1 induces p21 WAF1 and regulation of the cell cycle via direct activation of the JAK/Stat signaling pathway in a process requiring PKD2. Cell 109: 157-168.
2. Natarajan, E., et al. 2003. Co-expression of p16INK4a and Laminin α-5 γ-2 by microinvasive and superficial squamous cell carcinomas *in vivo* and by migrating wound and senescent keratinocytes in culture. Am. J. Pathol. 163: 477-491.
3. Lindstrom, M.S., et al. 2003. Myc and E2F1 induce p53 through p14ARF-independent mechanisms in human fibroblasts. Oncogene 22: 4993-5005.
4. Su, D., et al. 2010. Role of p38 MAPK pathway in BMP4-mediated Smad-dependent premature senescence in lung cancer cells. Biochem. J. 433: 333-343.
5. Li, H., et al. 2010. Transcriptional factor HBP1 targets P16(INK4A), upregulating its expression and consequently is involved in Ras-induced premature senescence. Oncogene 29: 5083-5094.
6. Spallarossa, P., et al. 2010. p38 MAPK and JNK antagonistically control senescence and cytoplasmic p16INK4A expression in doxorubicin-treated endothelial progenitor cells. PLoS ONE 5: e15583.
7. Wilson, B.G., et al. 2010. Epigenetic antagonism between polycomb and SWI/SNF complexes during oncogenic transformation. Cancer Cell 18: 316-328.
8. Zhang, W., et al. 2010. Comparison of the inhibitory effects of three transcriptional variants of CDKN2A in human lung cancer cell line A549. J. Exp. Clin. Cancer Res. 29: 74.
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