p19 INK4D (DCS-100): sc-56334



The Power to Question

BACKGROUND

The normal progression of cells through the cell cycle is under the control of the cyclin dependent protein kinases Cdk4 and Cdk6, which are subject to inhibition by the mitotic inhibitory protein, p16 INK4A. Isolated members of the p16 INK4A family have been designated p15 INK4B, p18 INK4C and p19 INK4D. p15 INK4B expression is upregulated approximately 30-fold in TGF β -treated human keratinocytes, suggesting that p15 INK4B may function as an effector of TGF β -mediated cell cycle arrest through inhibition of Cdk4 and Cdk6 kinases. The gene encoding p15 INK4B has been mapped to chromosome 9p21.3 at a position adjacent to the p16 INK4A gene, at a site of frequent chromosomal abnormality in human tumors. Two p16 INK4A-related proteins, p19 INK4D and p18 INK4C, specifically inhibit the kinase activities of Cdk4 and Cdk6 but do not affect those of cyclin E-Cdk2, cyclin A-Cdk2 or cyclin B-Cdc2 complexes. p19 INK4D is expressed at maximal level during S phase, while overexpression of p19 INK4D leads to G1 arrest.

CHROMOSOMAL LOCATION

Genetic locus: CDKN2D (human) mapping to 19p13.2.

SOURCE

p19 INK4D (DCS-100) is a mouse monoclonal antibody raised against full length p19 INK4D of human origin.

PRODUCT

Each vial contains 200 $\mu g \ lgG_1$ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

p19 INK4D (DCS-100) is available conjugated to agarose (sc-56334 AC), 500 $\mu g/0.25$ ml agarose in 1 ml, for IP; to HRP (sc-56334 HRP), 200 $\mu g/ml$, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-56334 PE), fluorescein (sc-56334 FITC), Alexa Fluor* 488 (sc-56334 AF488), Alexa Fluor* 546 (sc-56334 AF546), Alexa Fluor* 594 (sc-56334 AF594) or Alexa Fluor* 647 (sc-56334 AF647), 200 $\mu g/ml$, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor* 680 (sc-56334 AF680) or Alexa Fluor* 790 (sc-56334 AF790), 200 $\mu g/ml$, for Near-Infrared (NIR) WB, IF and FCM.

APPLICATIONS

p19 INK4D (DCS-100) is recommended for detection of p19 INK4D of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g 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 p19 INK4D siRNA (h): sc-36148, p19 INK4D shRNA Plasmid (h): sc-36148-SH and p19 INK4D shRNA (h) Lentiviral Particles: sc-36148-V.

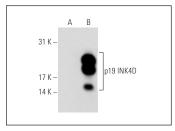
Molecular Weight of p19 INK4D: 19 kDa.

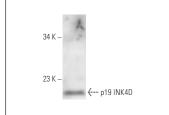
Positive Controls: p19 INK4D (h2): 293T Lysate: sc-174520 or Jurkat whole cell lysate: sc-2204.

STORAGE

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

DATA





p19 INK4D (DCS-100): sc-56334. Western blot analysis of p19 INK4D expression in non-transfected sc-117752 (**A**) and human p19 INK4D transfected: sc-174520 (**B**) 293T whole cell lysates.

p19 INK4D (DCS-100): sc-56334. Western blot analysis of p19 INK4D expression in Jurkat whole cell lysate.

SELECT PRODUCT CITATIONS

- Wu, W., et al. 2009. Antibody array analysis with label-based detection and resolution of protein size. Mol. Cell. Proteomics 8: 245-257.
- 2. Hervouet, E., et al. 2009. Dnmt3/transcription factor interactions as crucial players in targeted DNA methylation. Epigenetics 4: 487-499.
- 3. Por, E., et al. 2010. The cancer/testis antigen CAGE with oncogenic potential stimulates cell proliferation by up-regulating cyclins D1 and E in an AP-1- and E2F-dependent manner. J. Biol. Chem. 285: 14475-14485.
- 4. Jin, Y.J., et al. 2012. Macrophage inhibitory cytokine-1 stimulates proliferation of human umbilical vein endothelial cells by up-regulating cyclins D1 and E through the PI3K/Akt-, ERK-, and JNK-dependent AP-1 and E2F activation signaling pathways. Cell. Signal. 24: 1485-1495.
- 5. Jeanblanc, M., et al. 2012. Parallel pathways in RAF-induced senescence and conditions for its reversion. Oncogene 31: 3072-3085.
- Gogolin, S., et al. 2013. CDK4 inhibition restores G₁-S arrest in MYCNamplified neuroblastoma cells in the context of doxorubicin-induced DNA damage. Cell Cycle 12: 1091-1104.
- 7. Alessio, N., et al. 2019. The senescence-associated secretory phenotype (SASP) from mesenchymal stromal cells impairs growth of immortalized prostate cells but has no effect on metastatic prostatic cancer cells. Aging 11: 5817-5828.

RESEARCH USE

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

PROTOCOLS

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

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