SANTA CRUZ BIOTECHNOLOGY, INC.

p57 (H-91): sc-8298

SANTA CRUZ BIOTECHNOLOGY
The Power to Question

BACKGROUND

Cell cycle progression is regulated by a series of cyclin-dependent kinases that consist of catalytic subunits designated Cdks and activating subunits designated cyclins. Orderly progression through the cell cycle requires the activation and inactivation of different cyclin-Cdks at appropriate times. A series of proteins has been described that function as mitotic inhibitors. These include p21, the levels of which are elevated upon DNA damage in G_1 in a p53-dependent manner, p16 and p16-related inhibitors, designated p15, p18 and p19. A p21-related protein, p27, has been described as a negative regulator of G_1 progression and has been speculated to function as a possible mediator of TGFβ-induced G_1 arrest. A member of the p21/p27 family of mitotic inhibitory proteins has been designated p57. p57 is a potent, tight-binding cyclin-dependent inhibitor of several G_1 cyclin/Cdk complexes. Overexpression of p57 arrests cells in G_1. Unlike p21, p57 is not regulated by p53.

REFERENCES

1. Sherr, C.J. 1993. Mammalian G_1 cyclins. Cell 73: 1059-1065.
2. Xiong, Y., et al. 1993. p21 is a universal inhibitor of cyclin kinases. Nature 366: 701-704.

CHROMOSOMAL LOCATION

Genetic locus: CDKN1C (human) mapping to 11p15.4; Cdkn1c (mouse) mapping to 7 F5.

SOURCE

p57 (H-91) is a rabbit polyclonal antibody raised against amino acids 45-135 mapping within an internal region of p57 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.

APPLICATIONS

p57 (H-91) is recommended for detection of p57 of mouse, rat and 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 p57 siRNA (h): sc-35751, p57 siRNA (m): sc-37621, p57 shRNA Plasmid (h): sc-35751-SH, p57 shRNA Plasmid (m): sc-37621-SH, p57 shRNA (h) Lentiviral Particles: sc-35751-V and p57 shRNA (m) Lentiviral Particles: sc-37621-V.

Molecular Weight of p57: 57 kDa.

Positive Controls: A673 nuclear extract: sc-2128, Jurkat nuclear extract: sc-2132 or HeLa nuclear extract: sc-2120.

RESEARCH USE

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

SELECT PRODUCT CITATIONS

1. Carey, R.G., et al. 2002. Pituitary adenylate cyclase activating polypeptide anti-mitogenic signaling in cerebral cortical progenitors is regulated by $p57^{Kip2}$-dependent CDK2 activity. J. Neurosci. 22: 1583-1591.
2. Saravanamuthu, S.S., et al. 2009. Notch signaling is required for lateral induction of Jagged1 during FGF-induced lens fiber differentiation. Dev. Biol. 332: 166-176.
3. Bilodeau, S., et al. 2009. Distinct developmental roles of cell cycle inhibitors $p57^{Kip2}$ and $p27^{Kip1}$ distinguish pituitary progenitor cell cycle exit from cell cycle reentry of differentiated cells. Mol. Cell. Biol. 29: 1895-1908.
4. Haughian, J.M., et al. 2009. Protein kinase C α-dependent signaling mediates endometrial cancer cell growth and tumorigenesis. Int. J. Cancer 125: 2556-2564.
5. Adon, A.M., et al. 2010. Cdk2 and Cdk4 regulate the centrosome cycle and are critical mediators of centrosome amplification in p53-null cells. Mol. Cell. Biol. 30: 694-710.
6. Van Rechem, C., et al. 2010. Differential regulation of HIC1 target genes by CtBP and NuRD, via an acetylation/SUMOylation switch, in quiescent versus proliferating cells. Mol. Cell. Biol. 30: 4045-4059.
7. Ma, Y., et al. 2010. CDKN1C negatively regulates RNA polymerase II C-terminal domain phosphorylation in an E2F1-dependent manner. J. Biol. Chem. 285: 9813-9822.
8. Tury, A., et al. 2011. The cyclin-dependent kinase inhibitor $p57^{Kip2}$ regulates cell cycle exit, differentiation, and migration of embryonic cerebral cortical precursors. Cereb. Cortex 21: 1840-1856.
9. Gillespie, J.R., et al. 2011. Deletion of glycogen synthase kinase-3β in cartilage results in up-regulation of glycogen synthase kinase-3α protein expression. Endocrinology 152: 1755-1766.
10. Unek, G., et al. 2012. Immunolocalization of PCNA, Ki67, p27 and p57 in normal and dexamethasone-induced intrauterine growth restriction placental development in rat. Acta Histochem. 114: 31-40.
11. Joaquin, M., et al. 2012. The p57 CDKi integrates stress signals into cell-cycle progression to promote cell survival upon stress. EMBO J. 31: 2952-2964.

STORAGE

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