SANTA CRUZ BIOTECHNOLOGY, INC.

MDM2 (N-20): sc-813

SANTA CRUZ BIOTECHNOLOGY
The Power to Question

BACKGROUND

p53 is the most commonly mutated gene in human cancer identified to date. Expression of p53 leads to inhibition of cell growth by preventing progression of cells from G_1 to S phase of the cell cycle. Most importantly, p53 functions to cause arrest of cells in the G_1 phase of the cell cycle following any exposure of cells to DNA-damaging agents. The MDM2 (murine double minute-2) protein was initially identified as an oncogene in a murine transformation system. MDM2 functions to bind p53 and block p53-mediated transactivation of cotransfected reporter constructs. The MDM2 gene is amplified in a high percentage of human sarcomas that retain wildtype p53 and tumor cells that overexpress MDM2 can tolerate high levels of p53 expression. These findings argue that MDM2 overexpression represents at least one mechanism by which p53 function can be abrogated during tumorigenesis.

CHROMOSOMAL LOCATION

Genetic locus: MDM2 (human) mapping to 12q15.

SOURCE

MDM2 (N-20) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the N-terminus of MDM2 of human origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-813 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

MDM2 (N-20) is recommended for detection of MDM2 and MDM2 p60 cleavage product 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).

MDM2 (N-20) is also recommended for detection of MDM2 and MDM2 p60 cleavage product in additional species, including equine, canine, bovine, porcine and feline.

Suitable for use as control antibody for MDM2 siRNA (h): sc-29394, MDM2 shRNA Plasmid (h): sc-29394-SH and MDM2 shRNA (h) Lentiviral Particles: sc-29394-V.

Molecular Weight of MDM2: 90 kDa.

Molecular Weight of MDM2 cleavage product: 60 kDa.

Positive Controls: Jurkat whole cell lysate: sc-2204, U-2 OS cell lysate: sc-2295 or A-673 cell lysate: sc-2414.

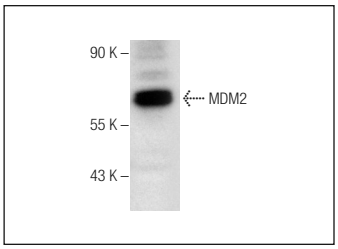
RESEARCH USE

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

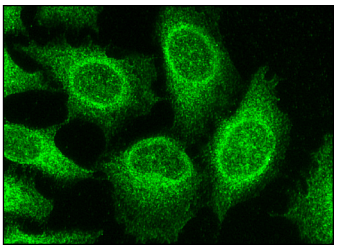
STORAGE

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

DATA



MDM2 (N-20): sc-813. Western blot analysis of MDM2 expression in Jurkat whole cell lysate.



MDM2 (N-20): sc-813. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic and nuclear localization.

SELECT PRODUCT CITATIONS

1. Maki, C.G., et al. 1999. Oligomerization is required for p53 to be efficiently ubiquitinated by MDM2. J. Biol. Chem. 274: 16531-16535.
2. Sarafraz-Yazdi, E., et al. 2010. Anticancer peptide PNC-27 adopts an HDM-2-binding conformation and kills cancer cells by binding to HDM-2 in their membranes. Proc. Natl. Acad. Sci. USA 107: 1918-1923.
3. Levin, V.A., et al. 2010. Different changes in protein and phosphoprotein levels result from serum starvation of high-grade glioma and adenocarcinoma cell lines. J. Proteome Res. 9: 179-191.
4. Xiong, X., et al. 2011. Ribosomal protein S27-like and S27 interplay with p53-MDM2 axis as a target, a substrate and a regulator. Oncogene 30: 1798-1811.
5. Kovacevic, Z., et al. 2011. The metastasis suppressor, N-myc downstream regulated gene 1 (NDRG1), upregulates p21 via p53-independent mechanisms. Carcinogenesis 32: 732-740.
6. Biderman, L., et al. 2012. MdmX is required for p53 interaction with and full induction of the Mdm2 promoter after cellular stress. Mol. Cell. Biol. 32: 1214-1225.
7. Fan, Y.H., et al. 2013. USP7 inhibitor P22077 inhibits neuroblastoma growth via inducing p53-mediated apoptosis. Cell Death Dis. 4: e867.
8. Halasi, M., et al. 2014. Proteasome inhibitors suppress the protein expression of mutant p53. Cell Cycle 13: 3202-3206.
9. Sun, F., et al. 2015. A novel prostate cancer therapeutic strategy using icaritin-activated arylhydrocarbon-receptor to co-target androgen receptor and its splice variants. Carcinogenesis 36: 757-768.