SANTA CRUZ BIOTECHNOLOGY, INC.

p-MDM2 (Ser 166): sc-293105

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. In response to ionization radiation, MDM2 may be phosphorylated on select amino acid residues, such as Thr 218.

REFERENCES

1. Kastan, M.B., et al. 1991. Participation of p53 protein in the cellular response to DNA damage. Cancer Res. 51: 6304-6311.
2. Kastan, M.B., et al. 1992. A mammalian cell cycle checkpoint pathway utilizing p53 and GADD 45 is defective in ataxia-telangiectasia. Cell 71: 587-597.
3. Oliner, J.D., et al. 1993. Oncoprotein MDM2 conceals the activation domain of tumor suppressor p53. Nature 362: 857-860.
4. Haines, D.S., et al. 1994. Physical and functional interaction between wildtype p53 and MDM2 proteins. Mol. Cell. Biol. 14: 1171-1178.
5. Chen, C.Y., et al. 1994. Interactions between p53 and MDM2 in a mammalian cell cycle checkpoint pathway. Proc. Natl. Acad. Sci. USA 91: 2684-2688.
6. Picksley, S.M., et al. 1994. Immunochemical analysis of the interaction of p53 with MDM2; fine mapping of the MDM2 binding site on p53 using synthetic peptides. Oncogene 9: 2523-2529.
7. Klein, C., et al. 2004. Targeting the p53-MDM2 interaction to treat cancer. Br. J. Cancer 91: 1415-1419.
8. Wang, Q., et al. 2008. Acidic domain is indispensable for MDM2 to negatively regulate the acetylation of p53. Biochem. Biophys. Res. Commun. 374: 437-441.
9. Brenkman, A.B., et al. 2008. MDM2 induces mono-ubiquitination of FOXO4. PLoS ONE 3: e2819.

CHROMOSOMAL LOCATION

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

SOURCE

p-MDM2 (Ser 166) is a rabbit polyclonal antibody raised against a short amino acid sequence containing Ser 166 phosphorylated MDM2 of human origin.

PRODUCT

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

APPLICATIONS

p-MDM2 (Ser 166) is recommended for detection of Ser 166 phosphorylated MDM2 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 immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500).

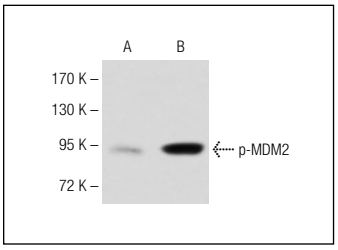
Suitable for use as control antibody for MDM2 siRNA (h): sc-29394, MDM2 shRNA Plasmid (h): sc-29394-SH and MDM2 shRNA (h) Lentiviral Particles: sc-29394-V.

Molecular Weight of p-MDM2 cleavage product: 60 kDa.

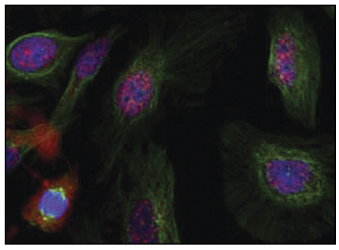
Molecular Weight of MDM2: 90 kDa.

Positive Controls: hydroxyurea treated 293 whole cell lysate.

DATA



p-MDM2 (Ser 166): sc-293105. Western blot analysis of MDM2 phosphorylation expression in untreated (**A**) and Hydroxyurea treated (**B**) 293 whole cell lysates.



p-MDM2 (Ser 166): sc-293105. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Zajkowicz, A. and Rusin, M. 2011. The activation of the p53 pathway by the AMP mimetic AICAR is reduced by inhibitors of the ATM or mTOR kinases. Mech. Ageing Dev. 132: 543-551.
2. Seal, S., et al. 2012. Vapor of volatile oils from *Litsea cubeba* seed induces apoptosis and causes cell cycle arrest in lung cancer cells. PLoS ONE 7: e47014.
3. Gravina, G.L., et al. 2015. Dual PI3K/mTOR inhibitor, XL765 (SAR245409), shows superior effects to sole PI3K [XL147 (SAR245408)] or mTOR [rapamycin] inhibition in prostate cancer cell models. Tumour Biol. E-published.

STORAGE

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

RESEARCH USE

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