SANTA CRUZ BIOTECHNOLOGY, INC.

p53 (C-19)-R: sc-1311-R

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

p53, a DNA-binding, oligomerization domain- and transcription activation domain-containing tumor suppressor, upregulates growth arrest and apoptosis-related genes in response to stress signals, thereby influencing programmed cell death, cell differentiation, and cell cycle control mechanisms. p53 localizes to the nucleus, yet can be chaperoned to the cytoplasm by the negative regulator, MDM2. MDM2 is an E3 ubiquitin ligase that is upregulated in the presence of active p53, where it poly-ubiquitinates p53 for proteasome targeting. p53 fluctuates between latent and active DNA-binding conformations and is differentially activated through posttranslational modifications, including phosphorylation and acetylation. Mutations in the DNA-binding domain (DBD) of p53, amino acids 110-286, can compromise energetically-favorable association with cis elements and are implicated in several human cancers.

CHROMOSOMAL LOCATION

Genetic locus: TP53 (human) mapping to 17p13.1; Trp53 (mouse) mapping to 11 B3.

SOURCE

p53 (C-19)-R is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of p53 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-1311 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

p53 (C-19)-R is recommended for detection of p53 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).

p53 (C-19)-R is also recommended for detection of p53 in additional species, including canine and bovine.

Suitable for use as control antibody for p53 siRNA (h): sc-29435, p53 siRNA (m): sc-29436, p53 shRNA Plasmid (h): sc-29435-SH, p53 shRNA Plasmid (m): sc-29436-SH, p53 shRNA (h) Lentiviral Particles: sc-29435-V and p53 shRNA (m) Lentiviral Particles: sc-29436-V.

Molecular Weight of p53: 53 kDa.

Positive Controls: p53 (m): 293T Lysate: sc-125766, A-431 whole cell lysate: sc-2201 or BT-20 cell lysate: sc-2223.

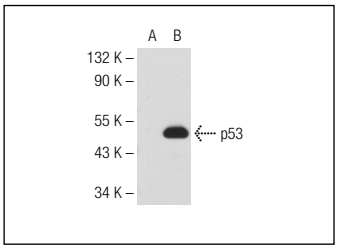
STORAGE

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

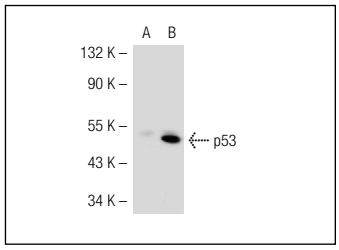
RESEARCH USE

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

DATA



p53 (C-19): sc-1311. Western blot analysis of p53 expression in non-transfected: sc-117752 (**A**) and mouse p53 transfected: sc-125766 (**B**) 293T whole cell lysates.



p53 (C-19): sc-1311. Western blot analysis of p53 expression in non-transfected: sc-117752 (**A**) and mouse p53 transfected: sc-125766 (**B**) 293T whole cell lysates.

SELECT PRODUCT CITATIONS

1. Gong, J.G., et al. 1999. The tyrosine kinase c-Abl regulates p73 in apoptotic response to cisplatin-induced DNA damage. Nature 399: 806-809.
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3. Park, K.S., et al. 2009. Inhibitory mechanism of ω-3 fatty acids in pancreatic inflammation and apoptosis. Ann. N.Y. Acad. Sci. 1171: 421-427.
4. Liu, W., et al. 2009. Disruption of estrogen receptor α-p53 interaction in breast tumors: a novel mechanism underlying the anti-tumor effect of radiation therapy. Breast Cancer Res. Treat. 115: 43-50.
5. Abedini, M.R., et al. 2010. Akt promotes chemoresistance in human ovarian cancer cells by modulating cisplatin-induced, p53-dependent ubiquitination of FLICE-like inhibitory protein. Oncogene 29: 11-25.
6. Lim, S.O., et al. 2011. Notch1 differentially regulates oncogenesis by wild-type p53 overexpression and p53 mutation in grade III hepatocellular carcinoma. Hepatology 53: 1352-1362.
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8. Yeghiazaryan, K., et al. 2011. Chromium-picolinate therapy in diabetes care: molecular and subcellular profiling revealed a necessity for individual outcome prediction, personalised treatment algorithms and new guidelines. Infect. Disord. Drug Targets 11: 188-195.
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