SANTA CRUZ BIOTECHNOLOGY, INC.

53BP1 (H-300): sc-22760

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The p53 binding proteins 53BP1 and 53BP2 (Bbp) bind to the central DNA-binding domain of wildtype p53, but do not bind mutant p53. The central DNA-binding domain of p53 is required for site-specific DNA binding and is frequently mutated in malignant tumors. Binding of 53BP1 to the L3 loop of p53 and of 53BP2 to the L2 loop of p53 confirms that the loop is dependent on p53 conformation. Site-specific binding also suggests that 53BP1 and 53BP2 are involved in p53-mediated tumor suppression. 53BP1 was isolated from H258 cells and is expressed in Jurkat cells in both the cytoplasm and the nucleus. The N-terminus of 53BP2 is localized to the cytoplasm, while the C-terminus might be localized in the nucleus. 53BP1 promotes cell proliferation by binding to p202, whereas 53BP2 induces cell death by binding to Bcl-2 and NFκB p65.

CHROMOSOMAL LOCATION

Genetic locus: TP53BP1 (human) mapping to 15q15.3; Trp53bp1 (mouse) mapping to 2 E5.

SOURCE

53BP1 (H-300) is a rabbit polyclonal antibody raised against amino acids 1-300 mapping at the N-terminus of 53BP1 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

53BP1 (H-300) is recommended for detection of 53BP1 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 53BP1 siRNA (h): sc-37455, 53BP1 siRNA (m): sc-37456, 53BP1 shRNA Plasmid (h): sc-37455-SH, 53BP1 shRNA Plasmid (m): sc-37456-SH, 53BP1 shRNA (h) Lentiviral Particles: sc-37455-V and 53BP1 shRNA (m) Lentiviral Particles: sc-37456-V.

Molecular Weight (predicted) of 53BP1: 214 kDa.

Molecular Weight (observed) of 53BP1: 245-460 kDa.

Positive Controls: Sol8 cell lysate: sc-2249, Ramos cell lysate: sc-2216 or HeLa+UV irradiated cell lysate: sc-2221.

STORAGE

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

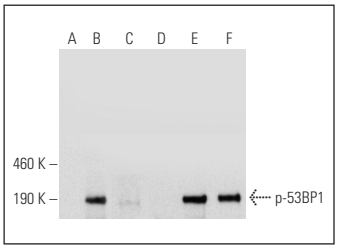
PROTOCOLS

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

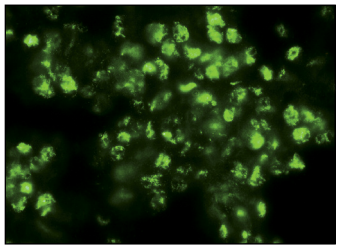
RESEARCH USE

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

DATA



Western blot analysis of 53BP1 phosphorylation in untreated (**A**,**D**), UV treated (**B**,**E**) and UV and lambda protein phosphatase (sc-200312A) treated (**C**,**F**) HeLa whole cell lysates. Antibodies tested include p-53BP1 (38.Ser 25): sc-135748 (**A**,**B**,**C**) and 53BP1 (H-300): sc-22760 (**D**,**E**,**F**).



53BP1 (H-300): sc-22760. Immunofluorescence staining of normal mouse kidney frozen section showing nuclear and cytoplasmic staining.

SELECT PRODUCT CITATIONS

1. Santos, C.R., et al. 2004. The vaccinia virus B1R kinase induces p53 downregulation by an Mdm2-dependent mechanism. Virology 328: 254-265.
2. Marteijn, J.A., et al. 2009. Nucleotide excision repair-induced H2A ubiquitination is dependent on MDC1 and RNF8 and reveals a universal DNA damage response. J. Cell Biol. 186: 835-847.
3. Hanot, M., et al. 2009. Membrane-dependent bystander effect contributes to amplification of the response to α-particle irradiation in targeted and nontargeted cells. Int. J. Radiat. Oncol. Biol. Phys. 75: 1247-1253.
4. Blazkova, H., et al. 2010. Bacterial intoxication evokes cellular senescence with persistent DNA damage and cytokine signalling. J. Cell. Mol. Med. 14: 357-367.
5. Khan, S.J., et al. 2010. Stress-induced senescence exaggerates postinjury neointimal formation in the old vasculature. Am. J. Physiol. Heart Circ. Physiol. 298: H66-H74.
6. Hicks, J.K., et al. 2010. Differential roles for DNA polymerases η, ζ, and REV1 in lesion bypass of intrastrand versus interstrand DNA cross-links. Mol. Cell. Biol. 30: 1217-1230.
7. Beck, H., et al. 2012. Cyclin-dependent kinase suppression by WEE1 kinase protects the genome through control of replication initiation and nucleotide consumption. Mol. Cell. Biol. 32: 4226-4236.
8. Kousholt, A.N., et al. 2012. CtIP-dependent DNA resection is required for DNA damage checkpoint maintenance but not initiation. J. Cell Biol. 197: 869-876.
9. Mosbech, A., et al. 2013. The deubiquitylating enzyme USP44 counteracts the DNA double-strand break response mediated by the RNF8 and RNF168 ubiquitin ligases. J. Biol. Chem. 288: 16579-16587.
10. Burrell, R.A., et al. 2013. Replication stress links structural and numerical cancer chromosomal instability. Nature 494: 492-496.