

# 53BP1 (6B3E10): sc-517281

## BACKGROUND

The p53 binding proteins 53BP1 and 53BP2 (Bbp) bind to the central DNA-binding domain of wild type 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 Bcl2 and NFκB p65.

## REFERENCES

- Iwabuchi, K., et al. 1994. Two cellular proteins that bind to wild-type but not mutant p53. *Proc. Natl. Acad. Sci. USA* 91: 6098-6102.
- Gorina, S. and Pavletich, N.P. 1996. Structure of the p53 tumor suppressor bound to the ankyrin and SH3 domains of 53BP2. *Science* 274: 1001-1005.
- Naumovski, L. and Cleary, M.L. 1996. The p53-binding protein 53BP2 also interacts with Bcl12 and impedes cell cycle progression at G<sub>2</sub>/M. *Mol. Cell. Biol.* 16: 3884-3892.

## CHROMOSOMAL LOCATION

Genetic locus: TP53BP1 (human) mapping to 15q15.3.

## SOURCE

53BP1 (6B3E10) is a mouse monoclonal antibody raised against a recombinant protein corresponding to amino acids 574-773 of 53BP1 of human origin.

## PRODUCT

Each vial contains 200 µg IgG<sub>1</sub> in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

## APPLICATIONS

53BP1 (6B3E10) is recommended for detection of 53BP1 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), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500), flow cytometry (1 µg per 1 x 10<sup>6</sup> cells) 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 shRNA Plasmid (h): sc-37455-SH and 53BP1 shRNA (h) Lentiviral Particles: sc-37455-V.

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

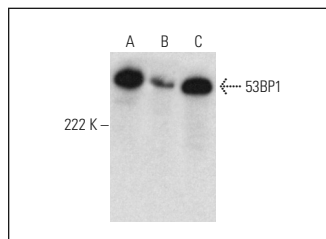
Molecular Weight (observed) of 53BP1: 245-460 kDa.

Positive Controls: Ramos cell lysate: sc-2216, HeLa whole cell lysate: sc-2200 or U-2 OS cell lysate: sc-2295.

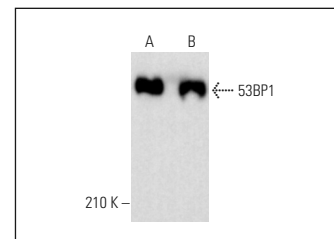
## STORAGE

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

## DATA



53BP1 (6B3E10): sc-517281. Western blot analysis of 53BP1 expression in Ramos (A), HeLa (B) and U-2 OS (C) whole cell lysates.



53BP1 (6B3E10): sc-517281. Western blot analysis of 53BP1 expression in IMR-32 (A) and Raji (B) whole cell lysates.

## SELECT PRODUCT CITATIONS

- Bhowmick, R., et al. 2019. The RIF1-PP1 axis controls abscission timing in human cells. *Curr. Biol.* 29: 1232-1242.e5.
- Sonneville, R., et al. 2019. TRAIIP drives replisome disassembly and mitotic DNA repair synthesis at sites of incomplete DNA replication. *Elife* 8: e48686.
- Chen, D., et al. 2022. BRCA1 deficiency specific base substitution mutagenesis is dependent on translesion synthesis and regulated by 53BP1. *Nat. Commun.* 13: 226.
- Jiang, B., et al. 2022. Mutations of the DNA repair gene PNKP in a patient with microcephaly, seizures, and developmental delay (MCSZ) presenting with a high-grade brain tumor. *Sci. Rep.* 12: 5386.
- Chakraborty, A., et al. 2023. Human DNA polymerase η promotes RNA-templated error-free repair of DNA double strand breaks. *J. Biol. Chem.* 299: 102991.
- Zhang, Z., et al. 2023. HDGFRP3 interaction with 53BP1 promotes DNA double-strand break repair. *Nucleic Acids Res.* 51: 2238-2256.
- Zhang, Q., et al. 2023. APE1 promotes non-homologous end joining by initiating DNA double-strand break formation and decreasing ubiquitination of artemis following oxidative genotoxic stress. *J. Transl. Med.* 21: 183.

## RESEARCH USE

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

## PROTOCOLS

See our web site at [www.scbt.com](http://www.scbt.com) for detailed protocols and support products.