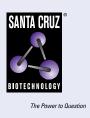
SANTA CRUZ BIOTECHNOLOGY, INC.

XRCC3 (10F1/6): sc-53471



BACKGROUND

The X-ray repair cross-complementing (XRCC) proteins are responsible for efficiently repairing and maintaining genetic stability following DNA base damage. These genes share sequence similarity with the yeast DNA repair protein Rad51. XRCC1 is a protein that facilitates the DNA base excision repair pathway by interacting with DNA ligase III and DNA polymerase to repair DNA single-strand breaks. XRCC2 and XRCC3 are both involved in maintaining chromosome stability during cell division. XRCC2 is required for efficient repair of DNA double-strand breaks by homologous recombination between sister chromatids, and XRCC3 interacts directly with Rad51 to cooperate with Rad51 during recombinational repair. XRCC4 is an accessory factor of DNA ligase IV that preferentially binds DNA with nicks or broken ends. XRCC4 binds to DNA ligase IV and enhances its joining activity, and it is also involved in V(D)J recombination. Any defect in one of the known components of the DNA repair/V(D)J recombination machinery (Ku-70, Ku-80, DNA-PK_{CS}, XRCC4 and DNA ligase IV) leads to abortion of the V(D)J rearrangement process and early block in both T and B cell maturation.

REFERENCES

- Tebbs, R.S., et al. 1995. Correction of chromosomal instability and sensitivity to diverse mutagens by a cloned cDNA of the XRCC3 DNA repair gene. Proc. Natl. Acad. Sci. USA 92: 6354-6358.
- Nash, R.A., et al. 1997. XRCC1 protein interacts with one of two distinct forms of DNA ligase III. Biochemistry 36: 5207-5211.
- Liu, N., et al. 1998. XRCC2 and XRCC3, new human Rad51-family members, promote chromosome stability and protect against DNA cross-links and other damages. Mol. Cell 1: 783-793.
- 4. Thacker, J. 1999. The role of homologous recombination processes in the repair of severe forms of DNA damage in mammalian cells. Biochimie 81: 77-85.
- Modesti, M., et al. 1999. DNA binding of XRCC4 protein is associated with V(D)J recombination but not with stimulation of DNA Ligase IV activity. EMBO J. 18: 2008-2018.
- Pierce, A.J., et al. 1999. XRCC3 promotes homology-directed repair of DNA damage in mammalian cells. Genes Dev. 13: 2633-2638.

CHROMOSOMAL LOCATION

Genetic locus: XRCC3 (human) mapping to 14q32.33.

SOURCE

XRCC3 (10F1/6) is a mouse monoclonal antibody raised against His-tagged recombinant XRCC3 of human origin.

PRODUCT

Each vial contains 200 μg IgG_1 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

RESEARCH USE

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

APPLICATIONS

XRCC3 (10F1/6) is recommended for detection of XRCC3 of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)].

Suitable for use as control antibody for XRCC3 siRNA (h): sc-37403, XRCC3 shRNA Plasmid (h): sc-37403-SH and XRCC3 shRNA (h) Lentiviral Particles: sc-37403-V.

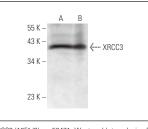
Molecular Weight of XRCC3: 40 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200.

RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker[™] Molecular Weight Standards: sc-2035, UltraCruz[®] Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml).

DATA



XRCC3 (10F1/6): sc-53471. Western blot analysis of XRCC3 expression in HeLa nuclear extract under reducing (**A**) and non-reducing conditions (**B**).

SELECT PRODUCT CITATIONS

- Oganesian, L. and Karlseder, J. 2011. Mammalian 5' C-rich telomeric overhangs are a mark of recombination-dependent telomere maintenance. Mol. Cell 42: 224-236.
- Oganesian, L. and Karlseder, J. 2013. 5' C-rich telomeric overhangs are an outcome of rapid telomere truncation events. DNA Repair 12: 238-245.
- Terasawa, M., et al. 2014. Canonical non-homologous end joining in mitosis induces genome instability and is suppressed by M-phase-specific phosphorylation of XRCC4. PLoS Genet. 10: e1004563.
- Somyajit, K., et al. 2021. Homology-directed repair protects the replicating genome from metabolic assaults. Dev. Cell 56: 461-477.e7.

STORAGE

Store at 4° C, **D0 NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.