SANTA CRUZ BIOTECHNOLOGY, INC.

XRCC4 (C-20): sc-8285

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-PKCS, 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.

CHROMOSOMAL LOCATION

Genetic locus: XRCC4 (human) mapping to 5q14.2; Xrcc4 (mouse) mapping to 13 C3.

SOURCE

XRCC4 (C-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the C-terminus of XRCC4 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-8285 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

XRCC4 (C-20) is recommended for detection of XRCC4 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).

XRCC4 (C-20) is also recommended for detection of XRCC4 in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for XRCC4 siRNA (h): sc-37405, XRCC4 siRNA (m): sc-37406, XRCC4 shRNA Plasmid (h): sc-37405-SH, XRCC4 shRNA Plasmid (m): sc-37406-SH, XRCC4 shRNA (h) Lentiviral Particles: sc-37405-V and XRCC4 shRNA (m) Lentiviral Particles: sc-37406-V.

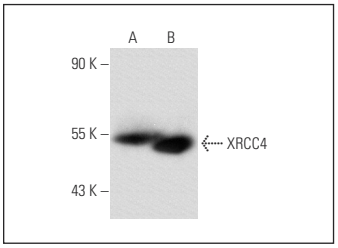
Molecular Weight of XRCC4: 55 kDa.

Positive Controls: MOLT-4 cell lysate: sc-2233, XRCC4 (h): 293T Lysate: sc-178146 or T-47D cell lysate: sc-2293.

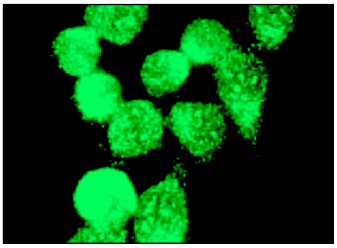
STORAGE

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

DATA



XRCC4 (C-20): sc-8285. Western blot analysis of XRCC4 expression in non-transfected: sc-117752 (**A**) and human XRCC4 transfected: sc-178146 (**B**) 293T whole cell lysates.



XRCC4 (C-20): sc-8285. Immunofluorescence staining of methanol-fixed HeLa cells showing nuclear and cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Baldeyron, C., et al. 2002. A single mutated BRCA1 allele leads to impaired fidelity of double strand break end-joining. Oncogene 21: 1401-1410.
2. Merel, P., et al. 2002. Absence of major defects in non-homologous DNA end joining in human breast cancer cell lines. Oncogene 21: 5654-5659.
3. Bertolini, L.R., et al. 2007. Transient depletion of Ku70 and Xrcc4 by RNAi as a means to manipulate the non-homologous end-joining pathway. J. Biotechnol. 128: 246-257.
4. Rubio, D., et al. 2008. Molecular characterization of spontaneous mesenchymal stem cell transformation. PLoS ONE 3: e1398.
5. Xie, A., et al. 2009. Role of mammalian Mre11 in classical and alternative nonhomologous end joining. Nat. Struct. Mol. Biol. 16: 814-818.
6. Bennardo, N., et al. 2009. Limiting the persistence of a chromosome break diminishes its mutagenic potential. PLoS Genet. 5: e1000683.
7. Tichy, E.D., et al. 2010. Mouse embryonic stem cells, but not somatic cells, predominantly use homologous recombination to repair double-strand DNA breaks. Stem Cells Dev. 19: 1699-1711.
8. Xing, Y., et al. 2011. DNA damage in embryonic stem cells caused by nanodiamonds. ACS Nano 5: 2376-2384.

RESEARCH USE

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