SANTA CRUZ BIOTECHNOLOGY, INC.

# MRE11 (C-16): sc-5859

SANTA CRUZ BIOTECHNOLOGY

*The Power to Question*

## BACKGROUND

Rad52 family members (Rad50, Rad51B/C/D, Rad52, Rad54 and MRE11) mediate DNA double-strand break repair (DSBR) for DNA damage that could otherwise cause cell death, mutation or neoplastic transformation. Rad51 (RECA, BRCC5) interacts with BRCA1 and BRCA2 to influence subcellular localization and cellular response to DNA damage. BRCA2 inactivation may be a key event leading to genomic instability and tumorigenesis from deregulation of Rad51. Rad52 forms a heptameric ring that binds single-stranded DNA ends and catalyzes DNA-DNA interaction necessary for the annealing of complementary strands. Rad52 can interact with Rad51. MRE11 (meiotic recombination 11, ATLD, HNGS1) is a nuclear 3'-5' exonuclease/endonuclease that associates with RAD50 and influences homologous recombination, telomere length maintenance, and DNA double-strand break repair. MRE11 is most abundant in proliferating tissues.

## CHROMOSOMAL LOCATION

Genetic locus: MRE11A (human) mapping to 11q21; Mre11a (mouse) mapping to 9 A2.

## SOURCE

MRE11 (C-16) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the C-terminus of MRE11 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-5859 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

## APPLICATIONS

MRE11 (C-16) is recommended for detection of MRE11 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 MRE11 siRNA (h): sc-37395, MRE11 siRNA (m): sc-37396, MRE11 shRNA Plasmid (h): sc-37395-SH, MRE11 shRNA Plasmid (m): sc-37396-SH, MRE11 shRNA (h) Lentiviral Particles: sc-37395-V and MRE11 shRNA (m) Lentiviral Particles: sc-37396-V.

Molecular Weight of MRE11: 80 kDa.

Positive Controls: K-562 whole cell lysate: sc-2203, HeLa whole cell lysate: sc-2200 or Jurkat whole cell lysate: sc-2204.

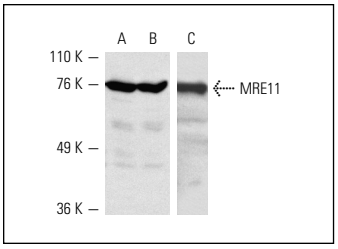
## STORAGE

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

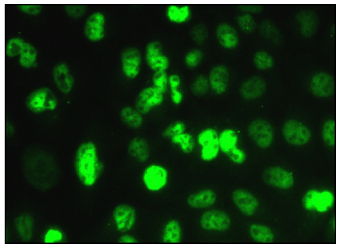
## RESEARCH USE

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

## DATA




MRE11 (C-16): sc-5859. Western blot analysis of MRE11 expression in K-562 (**A**), HeLa (**B**) and Jurkat (**C**) whole cell lysates.



MRE11 (C-16): sc-5859. Immunofluorescence staining of formalin-fixed HeLa cells showing nuclear localization. Kindly provided by Yang Xiang, Ph.D., Division of Newborn Medicine, Boston Children's Hospital, Cell Biology Department, Harvard Medical School.

## SELECT PRODUCT CITATIONS


1. Rapp, A., et al. 2004. After double-strand break induction by UV-A, homologous recombination and nonhomologous end joining cooperate at the same DSB if both systems are available. J. Cell Sci. 117: 4935-4945.
2. Bentley, J., et al. 2004. DNA double strand break repair in human bladder cancer is error prone and involves microhomology-associated end-joining. Nucleic Acids Res. 32: 5249-5529.
3. Singh, S., et al. 2009. Modification in the expression of Mre11/Rad50/Nbs1 complex in low dose irradiated human lymphocytes. Dose Response 7: 193-207.
4. Caslini, C., et al. 2009. MLL associates with telomeres and regulates telo-meric repeat-containing RNA transcription. Mol. Cell. Biol. 29: 4519-4526.
5. Li, B., et al. 2011. Depletion of Ku70/80 reduces the levels of extrachromosomal telomeric circles and inhibits proliferation of ALT cells. Aging 3: 395-406.
6. Gannon, A.M., et al. 2012. MutSβ and histone deacetylase complexes promote expansions of trinucleotide repeats in human cells. Nucleic Acids Res. 40: 10324-10333.
7. Sousa, M.M., et al. 2013. An inverse switch in DNA base excision and strand break repair contributes to melphalan resistance in multiple myeloma cells. PLoS ONE 8: e55493.
8. Chaudhury, I., et al. 2014. FANCD2-controlled chromatin access of the Fanconi-associated nuclease FAN1 is crucial for the recovery of stalled replication forks. Mol. Cell. Biol. 34: 3939-3954.