Rad51C (2H11): sc-56214



The Power to Question

BACKGROUND

Rad52 family members (Rad50, Rad51B/C/D, Rad52, Rad54, MRE11) mediate DNA double-strand break repair (DSBR) for DNA damage that otherwise could 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. Rad54A of the DEAD-like helicase superfamily binds to double-strand DNA and induces a DNA topological change, which is thought to facilitate homologous DNA pairing and stimulate DNA recombination. Rad54B of the DEAD-like helicase superfamily binds to double-stranded DNA and displays ATPase activity in the presence of DNA. Rad54B is abundant in testis and spleen, and mutations of this gene occur in primary lymphoma and colon cancer. 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.

REFERENCES

- Tsukamoto, Y., et al. 1996. Effects of mutations of Rad50, Rad51, Rad52, and related genes on illegitimate recombination in Saccharomyces cerevisiae. Genetics 142: 383-391.
- 2. Zhong, Q., et al. 2002. Deficient nonhomologous end-joining activity in cell-free extracts from BRCA1-null fibroblasts. Cancer Res. 62: 3966-3970.

CHROMOSOMAL LOCATION

Genetic locus: RAD51C (human) mapping to 17q22; Rad51c (mouse) mapping to 11 C.

SOURCE

Rad51C (2H11) is a mouse monoclonal antibody raised against Rad51C of human origin.

PRODUCT

Each vial contains 200 μ g IgG₁ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Rad51C (2H11) is available conjugated to agarose (sc-56214 AC), 500 μ g/ 0.25 ml agarose in 1 ml, for IP; to HRP (sc-56214 HRP), 200 μ g/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-56214 PE), fluorescein (sc-56214 FITC), Alexa Fluor* 488 (sc-56214 AF488), Alexa Fluor* 546 (sc-56214 AF546), Alexa Fluor* 594 (sc-56214 AF594) or Alexa Fluor* 647 (sc-56214 AF647), 200 μ g/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor* 680 (sc-56214 AF680) or Alexa Fluor* 790 (sc-56214 AF790), 200 μ g/ml, for Near-Infrared (NIR) WB, IF and FCM.

STORAGE

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

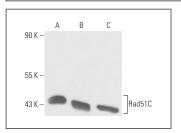
APPLICATIONS

Rad51C (2H11) is recommended for detection of Rad51C of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)]; non cross-reactive with Rad51B, Rad51D, Rad51, XRCC2, or XRCC3.

Suitable for use as control antibody for Rad51C siRNA (h): sc-45956, Rad51C siRNA (m): sc-45957, Rad51C shRNA Plasmid (h): sc-45956-SH, Rad51C shRNA Plasmid (m): sc-45957-SH, Rad51C shRNA (h) Lentiviral Particles: sc-45956-V and Rad51C shRNA (m) Lentiviral Particles: sc-45957-V.

Molecular Weight of Rad51C: 42 kDa.

DATA



Rad51C (2H11): sc-56214. Western blot analysis of Rad51C expression in HEK293 (**A**), ES-2 (**B**) and PCEP-4 (**C**) whole cell lysates.

SELECT PRODUCT CITATIONS

- 1. Alayev, A., et al. 2016. Estrogen induces Rad51C expression and localization to sites of DNA damage. Cell Cycle 15: 3230-3239.
- Dawson, L.M., et al. 2019. A dominant Rad51C pathogenic splicing variant predisposes to breast and ovarian cancer in the Newfoundland population due to founder effect. Mol. Genet. Genomic Med. 8: e1070.
- Nesic, K., et al. 2021. Acquired Rad51C promoter methylation loss causes PARP inhibitor resistance in high-grade serous ovarian carcinoma. Cancer Res. 81: 4709-4722.
- Durinikova, E., et al. 2022. Targeting the DNA damage response pathways and replication stress in colorectal cancer. Clin. Cancer Res. 28: 3874-3889.
- Dixit, S., et al. 2024. RTEL1 helicase counteracts RAD51-mediated homologous recombination and fork reversal to safeguard replicating genomes. Cell Rep. 43: 114594.
- Sogari, A., et al. 2024. Tolerance to colibactin correlates with homologous recombination proficiency and resistance to irinotecan in colorectal cancer cells. Cell Rep. Med. 5: 101376.

RESEARCH USE

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

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