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RAD51C Antibody (Center) Blocking Peptide

Synthetic peptide
Catalog # BP14962c

Specification

RAD51C Antibody (Center) Blocking Peptide - Product Information

Primary Accession O43502

RAD51C Antibody (Center) Blocking Peptide - Additional Information

Gene ID 5889

Other Names
DNA repair protein RAD51 homolog 3, R51H3, RAD51 homolog C, RAD51-like protein 2, RAD51C, RAD51L2

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

RAD51C Antibody (Center) Blocking Peptide - Protein Information

Name RAD51C

Synonyms RAD51L2

Function
Essential for the homologous recombination (HR) pathway of DNA repair. Involved in the homologous recombination repair (HRR) pathway of double-stranded DNA breaks arising during DNA replication or induced by DNA-damaging agents. Part of the RAD21 paralog protein complexes BCDX2 and CX3

RAD51C Antibody (Center) Blocking Peptide - Background

This gene is a member of the RAD51 family of relatedgenes, which encode strand-transfer proteins thought to be involvedin recombinational repair of damaged DNA and in meioticrecombination. This gene product interacts with two other DNArepair proteins, encoded by RAD51B and XRCC3, but not with itself.The protein copurifies with XRCC3 protein in a complex, reflectingtheir endogenous association and suggesting a cooperative roleduring recombinational repair. This gene is one of four localizedto a region of chromosome 17q23 where amplification occursfrequently in breast tumors. Overexpression of the four genesduring amplification has been observed and suggests a possible rolein tumor progression. Alternative splicing has been observed forthis gene and two variants encoding different isoforms have beenidentified.

RAD51C Antibody (Center) Blocking Peptide - References

Zheng, Y., et al. Breast Cancer Res. Treat. (2010) In press :Briggs, F.B., et al. Am. J. Epidemiol. 172(2):217-224(2010)Monsees, G.M., et al. Breast Cancer Res. Treat. (2010) In press :Meindl, A., et al. Nat. Genet. 42(5):410-414(2010)Vaz, F., et al. Nat. Genet. 42(5):406-409(2010)

which act at different stages of the BRCA1-BRCA2-dependent HR pathway. Upon DNA damage, BCDX2 seems to act downstream of BRCA2 recruitment and upstream of RAD51 recruitment; CX3 seems to act downstream of RAD51 recruitment; both complexes bind predominantly to the intersection of the four duplex arms of the Holliday junction (HJ) and to junction of replication forks. The BCDX2 complex was originally reported to bind single-stranded DNA, single- stranded gaps in duplex DNA and specifically to nicks in duplex DNA. The BCDX2 subcomplex RAD51B:RAD51C exhibits single-stranded DNA- dependent ATPase activity suggesting an involvement in early stages of the HR pathway. Involved in RAD51 foci formation in response to DNA damage suggesting an involvement in early stages of HR probably in the invasion step. Has an early function in DNA repair in facilitating phosphorylation of the checkpoint kinase CHEK2 and thereby transduction of the damage signal, leading to cell cycle arrest and HR activation. Participates in branch migration and HJ resolution and thus is important for processing HR intermediates late in the DNA repair process; the function may be linked to the CX3 complex. Part of a PALB2-scaffolded HR complex containing BRCA2 and which is thought to play a role in DNA repair by HR. Protects RAD51 from ubiquitin-mediated degradation that is enhanced following DNA damage. Plays a role in regulating mitochondrial DNA copy number under conditions of oxidative stress in the presence of RAD51 and XRCC3. Contributes to DNA cross- link resistance, sister chromatid cohesion and genomic stability. Involved in maintaining centrosome number in mitosis.

Cellular Location
Nucleus. Cytoplasm. Cytoplasm, perinuclear region. Mitochondrion. Note=DNA damage induces an increase in nuclear levels. Accumulates in DNA damage induced nuclear foci or RAD51C foci which is formed during the S or G2 phase of cell cycle. Accumulation at DNA lesions requires the presence of NBN/NBS1, ATM and RPA

Tissue Location
Expressed in a variety of tissues, with highest expression in testis, heart muscle, spleen and prostate

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RAD51C Antibody (Center) Blocking Peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides