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SMC1A Antibody (N-term) Blocking peptide
Synthetic peptide
Catalog # BP13544a

Specification

SMC1A Antibody (N-term) Blocking peptide - Product Information

Primary Accession Q14683

SMC1A Antibody (N-term) Blocking peptide - Additional Information

Gene ID 8243

Other Names
Structural maintenance of chromosomes protein 1A, SMC protein 1A, SMC-1-alpha, SMC-1A, Sb18, SMC1A, DXS423E, KIAA0178, SB18, SMC1, SMC1L1

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP13544a was selected from the N-term region of SMC1A. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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.

SMC1A Antibody (N-term) Blocking peptide - Protein Information

Name SMC1A

Synonyms DXS423E, KIAA0178, SB1.8, SMC1, SMC1L1

Function
Involved in chromosome cohesion during cell cycle and in DNA repair. Central component of cohesin complex. The cohesin complex is required for the cohesion of sister chromatids after DNA replication. The cohesin complex apparently forms a large proteinaceous ring within which sister chromatids can be trapped. At anaphase, the complex is cleaved and dissociates from chromatin, allowing sister chromatids to segregate. The cohesin complex may also play a role in spindle pole assembly during mitosis. Involved in DNA repair via its interaction with BRCA1 and its related phosphorylation by ATM, or via its phosphorylation by ATR. Works as a downstream effector both in the ATM/NBS1 branch and in the ATR/MSH2 branch of S-phase checkpoint.

Cellular Location

Nucleus. Chromosome. Chromosome, centromere, kinetochore. Note=Associates with chromatin. Before prophase it is scattered along chromosome arms. During prophase, most of cohesin complexes dissociate from chromatin probably because of phosphorylation by PLK, except at centromeres, where cohesin complexes remain. At anaphase, the RAD21 subunit of the cohesin complex is cleaved, leading to the dissociation of the complex from chromosomes, allowing chromosome separation. In germ cells, cohesin complex dissociates from chromatin at prophase I, and may be replaced by a meiosis-specific cohesin complex. The phosphorylated form on Ser-957 and Ser-966 associates with chromatin during G1/S/G2 phases but not during M phase, suggesting that phosphorylation does not regulate cohesin function. Integral component of the functional centromere- kinetochore complex at the kinetochore region during mitosis

SMC1A Antibody (N-term) Blocking peptide - Protocols

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

- Blocking Peptides

SMC1A Antibody (N-term) Blocking peptide - Images

SMC1A Antibody (N-term) Blocking peptide - Background

Proper cohesion of sister chromatids is a prerequisite forthe correct segregation of chromosomes during cell division. Thecohesin multiprotein complex is required for sister chromatidcohesion. This complex is composed partly of two structuralmaintenance of chromosomes (SMC) proteins, SMC3 and either SMC1L2or the protein encoded by this gene. Most of the cohesin complexesdissociate from the chromosomes before mitosis, although thosecomplexes at the kinetochore remain. Therefore, the encoded proteinis thought to be an important part of functional kinetochores. Inaddition, this protein interacts with BRCA1 and is phosphorylatedby ATM, indicating a potential role for this protein in DNA repair.This gene, which belongs to the SMC gene family, is located in anarea of the X-chromosome that escapes X inactivation. [provided byRefSeq].

SMC1A Antibody (N-term) Blocking peptide - References

Limongelli, G., et al. Am. J. Med. Genet. A 152A (8), 2127-2129 (2010) :Homme, C., et al. Oncol. Rep. 24(1):47-56(2010)Pie, J., et al. Am. J. Med. Genet. A 152A (4), 924-929 (2010) :Wong, R.W. Cell Cycle 9(1):198-200(2010)Liu, J., et al. Hum. Mutat. 30(11):1535-1542(2009)