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SNF2L Antibody (C-term) Blocking Peptide
Synthetic peptide
Catalog # BP9257b

Specification

SNF2L Antibody (C-term) Blocking Peptide - Product Information

Primary Accession P28370

SNF2L Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 6594

Other Names
Probable global transcription activator SNF2L1, 364-, ATP-dependent helicase SMARCA1, Nucleosome-remodeling factor subunit SNF2L, SWI/SNF-related matrix-associated actin-dependent regulator of chromatin subfamily A member 1, SMARCA1, SNF2L, SNF2L1

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP9257b was selected from the C-term region of human SNF2L. 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.

SNF2L Antibody (C-term) Blocking Peptide - Protein Information

Name SMARCA1 (HGNC:11097)

Synonyms SNF2L, SNF2L1

Function
[Isoform 1]: ATPase that possesses intrinsic ATP-dependent chromatin-remodeling activity (PubMed:14609955, PubMed:15310751, PubMed:15640247, PubMed:28801535). ATPase activity is substrate- dependent, and is increased when nucleosomes are the substrate, but is also catalytically active when DNA alone is the substrate (PubMed:14609955, PubMed:<a

href="http://www.uniprot.org/citations/15310751" target="_blank">15310751, PubMed:15640247). Catalytic subunit of ISWI chromatin-remodeling complexes, which form ordered nucleosome arrays on chromatin and facilitate access to DNA during DNA-templated processes such as DNA replication, transcription, and repair (PubMed:14609955, PubMed:15310751, PubMed:15640247, PubMed:28801535). Within the ISWI chromatin-remodeling complexes, slides edge- and center-positioned histone octamers away from their original location on the DNA template (PubMed:28801535). Catalytic activity and histone octamer sliding propensity is regulated and determined by components of the ISWI chromatin-remodeling complexes (PubMed:28801535). The BAZ1A-, BAZ1B-, BAZ2A- and BAZ2B-containing ISWI chromatin-remodeling complexes regulate the spacing of nucleosomes along the chromatin and have the ability to slide mononucleosomes to the center of a DNA template (PubMed:28801535). The CECR2- and RSF1-containing ISWI chromatin- remodeling complexes do not have the ability to slide mononucleosomes to the center of a DNA template (PubMed:28801535). Within the NURF-1 and CERF-1 ISWI chromatin remodeling complexes, nucleosomes are the preferred substrate for its ATPase activity (PubMed:14609955, PubMed:15640247). Within the NURF-1 ISWI chromatin-remodeling complex, binds to the promoters of En1 and En2 to positively regulate their expression and promote brain development (PubMed:14609955). May promote neurite outgrowth (PubMed:14609955). May be involved in the development of luteal cells (PubMed:16740656). Facilitates nucleosome assembly during DNA replication, ensuring replication fork progression and genomic stability by preventing replication stress and nascent DNA gaps (PubMed:39413208).

Cellular Location
Nucleus. Chromosome

Tissue Location
[Isoform 1]: Expressed in lung, breast, kidney, ovary, skeletal muscle and brain.

SNF2L Antibody (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

SNF2L Antibody (C-term) Blocking Peptide - Images

SNF2L Antibody (C-term) Blocking Peptide - Background

SNF2L encodes a member of the SWI/SNF family of proteins. Members of this family have helicase and ATPase activities and are thought to regulate transcription of certain genes by altering the chromatin structure around those genes.

SNF2L Antibody (C-term) Blocking Peptide - References

Ye,Y., et.al., Mol. Cancer Res. 7 (12), 1984-1999 (2009)Xia,Y., et.al., Biochem. Biophys. Res.

Commun. 368 (2), 438-444 (2008)Lazzaro,M.A., et.al., BMC Med. Genet. 9, 11 (2008)