

SULT2A Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP2603b

Specification

SULT2A Antibody (C-term) Blocking Peptide - Product Information

Primary Accession [O06520](#)
Other Accession [NP_003158](#)

SULT2A Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 6822

Other Names

Bile salt sulfotransferase, Dehydroepiandrosterone sulfotransferase, DHEA-ST, Hydroxysteroid Sulfotransferase, HST, ST2, ST2A3, Sulfotransferase 2A1, ST2A1, SULT2A1, HST, STD

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP2603b](/product/products/AP2603b) was selected from the C-term region of human SULT2A. 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.

SULT2A Antibody (C-term) Blocking Peptide - Protein Information

Name SULT2A1

Synonyms HST, STD

Function

Sulfotransferase that utilizes 3'-phospho-5'-adenylyl sulfate (PAPS) as sulfonate donor to catalyze the sulfonation of steroids and bile acids in the liver and adrenal glands. Mediates the sulfation of a wide range of steroids and sterols, including pregnenolone, androsterone, DHEA, bile acids, cholesterol and as well many xenobiotics that contain alcohol and phenol functional groups (PubMed: [14573603](http://www.uniprot.org/citations/14573603), PubMed: [18042734](http://www.uniprot.org/citations/18042734), PubMed: [19589875](http://www.uniprot.org/citations/19589875), PubMed: [21187059](http://www.uniprot.org/citations/21187059),

PubMed: 2268288, PubMed: 29671343, PubMed: 7678732, PubMed: 7854148. Sulfonation increases the water solubility of most compounds, and therefore their renal excretion, but it can also result in bioactivation to form active metabolites. Plays an important role in maintaining steroid and lipid homeostasis (PubMed: 14573603, PubMed: 19589875, PubMed: 21187059). Plays a key role in bile acid metabolism (PubMed: 2268288). In addition, catalyzes the metabolic activation of potent carcinogenic polycyclic arylmethanols (By similarity).

Cellular Location

Cytoplasm.

Tissue Location

Liver, adrenal and at lower level in the kidney. Is present in human fetus in higher level in the adrenal than the liver and the kidney

SULT2A Antibody (C-term) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

SULT2A Antibody (C-term) Blocking Peptide - Images

SULT2A Antibody (C-term) Blocking Peptide - Background

One of the major roles of the sulfotransferases (ST) in the metabolism of drugs and endogenous compounds is the conversion of these substances into more hydrophilic water-soluble sulfate conjugates that can be easily excreted. Sulfation may also play a regulatory role for many endogenous compounds, such as steroids and neurotransmitters, by altering the biologic properties of these compounds. Otterness et al. (1992), Kong et al. (1992), and Comer et al. (1993) reported the cloning of cDNAs encoding liver dehydroepiandrosterone (DHEA) sulfotransferase. The predicted protein has 285 amino acids. Although Northern blot analysis of human liver RNA detected transcripts of 3 different sizes, Southern blot analysis of human DNA suggested that only 1 gene is present in the genome. This gene has an important role in the sulfation of both bile acids and steroids in the liver and adrenals. The human adrenal form of this enzyme is physically, immunologically, and kinetically similar, perhaps identical, to the liver form. Dehydroepiandrosterone sulfate is quantitatively one of the major steroids secreted from the adrenal cortex.

SULT2A Antibody (C-term) Blocking Peptide - References

Otterness, D. M., et al. *Molec. Pharm.* 41: 865-872 (1992). Kong, A.-N. T., et al. *Biochem. Biophys. Res. Commun.* 187: 448-454 (1992). Comer, K. A., et al. *Biochem. J.* 289: 233-240 (1993).