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SULT1B1 Antibody (N-term) Blocking Peptide
Synthetic peptide
Catalog # BP2605a

Specification

SULT1B1 Antibody (N-term) Blocking Peptide - Product Information

Primary Accession	O43704
Other Accession	NP_055280

SULT1B1 Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 27284

Other Names
Sulfotransferase family cytosolic 1B member 1, ST1B1, Sulfotransferase 1B1, 282-, Sulfotransferase 1B2, ST1B2, Thyroid hormone sulfotransferase, SULT1B1, ST1B2, SULT1B2

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP2605a was selected from the N-term region of human SULT1B1. 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.

SULT1B1 Antibody (N-term) Blocking Peptide - Protein Information

Name SULT1B1

Synonyms ST1B2 {ECO:0000303|PubMed:9443824}, SULT

Function
Sulfotransferase that utilizes 3'-phospho-5'-adenylyl sulfate (PAPS) as sulfonate donor to catalyze the sulfate conjugation of dopamine, small phenols such as 1-naphthol and p-nitrophenol and thyroid hormones, including 3,3'-diiodothyronine, triidothyronine (T3) and reverse triiodothyronine (rT3) (PubMed:28084139, PubMed:9443824, PubMed:9463486). May play a role in gut microbiota-host metabolic interaction. O-sulfonates 4-ethylphenol (4-EP), a dietary tyrosine- derived metabolite produced by gut bacteria.

The product 4-EPS crosses the blood-brain barrier and may negatively regulate oligodendrocyte maturation and myelination, affecting the functional connectivity of different brain regions associated with the limbic system (PubMed:35165440).

Cellular Location
Cytoplasm

Tissue Location
Highly expressed in the liver, peripheral blood leukocytes, colon (mucosal lining), small intestine (jejunum) and spleen. A lesser expression was observed in the lung, placenta and thymus.

SULT1B1 Antibody (N-term) Blocking Peptide - Protocols

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

- Blocking Peptides

SULT1B1 Antibody (N-term) Blocking Peptide - Images

SULT1B1 Antibody (N-term) Blocking Peptide - Background

Cytosolic sulfotransferase (ST) enzymes catalyze the sulfate conjugation of many drugs, xenobiotic compounds, hormones, and neurotransmitters. Sulfotransferases such as SULT1B1 catalyze the biotransformation of a large number of endogenous compounds such as neurotransmitters, steroids, bile acids, and thyroid hormones, as well as drugs and xenobiotics. SULT1B1 contains a binding site for the sulfate donor, 3-prime-phosphoadenosine 5-prime-phosphosulfate, and a cysteine residue conserved in the ST1 gene family of sulfotransferases. SULT1B1 shares between 51.5% and 56.3% amino acid sequence identity with members of the phenol sulfotransferase gene family. Northern blot analysis detects 4 SULT1B1 transcripts between 79 kb and 13 kb expressed at highest levels in liver, peripheral blood leukocytes, colon, small intestine, and spleen. Lower expression is detected in lung, placenta, and thymus, and no expression is detected in other tissues. Recombinant rat and human SULT1B1, expressed in E. coli, catalyze sulfation of p-nitrophenol, 3,3-prime,5-triiodothyronine (T3), and dopamine, but not of beta-estradiol and dehydroepiandrosterone. SULT1B1 shows higher affinities for formation of T3 sulfate than do the phenol sulfotransferases ST1A3 (SULT1A1) or ST1A5. Bacterially-expressed SULT1B1 sulfates small phenols such as 1-naphthol and p-nitrophenol and thyroid hormones, including 3,3-prime-diiodothyronine, triiodothyronine, reverse triiodothyronine, and thyroxine. No activity is detected against several sterols or dopamine.

SULT1B1 Antibody (N-term) Blocking Peptide - References

Fujita, K., et al., J. Biochem. 122: 1052-1061 (1997).Wang, J., et al., Molec. Pharm. 53: 274-282 (1998).