

**SULT2A1 Antibody (N-term)**  
**Affinity Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP16646a****Specification**

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**SULT2A1 Antibody (N-term) - Product Information**

Application	WB,E
Primary Accession	<a href="#">Q06520</a>
Other Accession	<a href="#">P52842</a> , <a href="#">NP_003158.2</a>
Reactivity	Human
Predicted	Monkey
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	33780
Antigen Region	2-30

**SULT2A1 Antibody (N-term) - 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**

This SULT2A1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 2-30 amino acids from the N-terminal region of human SULT2A1.

**Dilution**

WB~~1:1000

**Format**

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

**Storage**

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**

SULT2A1 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

**SULT2A1 Antibody (N-term) - 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:[7678732](#), PubMed:[2268288](#), PubMed:[14573603](#), PubMed:[18042734](#), PubMed:[19589875](#), PubMed:[21187059](#), PubMed:[29671343](#), 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:[21187059](#), PubMed:[19589875](#), PubMed:[14573603](#)). 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

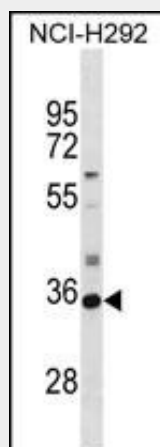
Liver, adrenal and at lower level in the kidney. Is present in human fetus in higher level in the adrenal than the liver and the kidney

## SULT2A1 Antibody (N-term) - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

## SULT2A1 Antibody (N-term) - Images



SULT2A1 Antibody (N-term) (Cat. #AP16646a) western blot analysis in NCI-H292 cell line lysates (35ug/lane). This demonstrates the SULT2A1 antibody detected the SULT2A1 protein (arrow).

## SULT2A1 Antibody (N-term) - Background

This gene encodes a member of the sulfotransferase family. Sulfotransferases aid in the metabolism of drugs and endogenous compounds by converting these substances into more hydrophilic water-soluble sulfate conjugates that can be easily excreted. This protein catalyzes the sulfation of steroids and bile acids in the liver and adrenal glands, and may have a role in the inherited adrenal androgen excess in women with polycystic ovary syndrome.

#### **SULT2A1 Antibody (N-term) - References**

Huang, J., et al. Xenobiotica 40(3):184-194(2010)  
Li, J., et al. Breast Cancer Res. 12 (2), R19 (2010) :  
Senggunprai, L., et al. Drug Metab. Dispos. 37(8):1711-1717(2009)  
Chakrabarti, B., et al. Autism Res 2(3):157-177(2009)  
Saito, A., et al. J. Hum. Genet. 54(6):317-323(2009)