10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

CYP26A1 Antibody (Center) Blocking Peptide

Synthetic peptide
Catalog # BP2754c

Specification

CYP26A1 Antibody (Center) Blocking Peptide - Product Information

Primary Accession	O43174
Other Accession	Q5VXI0

CYP26A1 Antibody (Center) Blocking Peptide - Additional Information

Gene ID 1592

Other Names
Cytochrome P450 26A1, 114--, Cytochrome P450 retinoic acid-inactivating 1, Cytochrome P450RAI, hP450RAI, Retinoic acid 4-hydroxylase, Retinoic acid-metabolizing cytochrome, CYP26A1, CYP26, P450RAI1

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP2754c was selected from the Center region of human CYP26A1. 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.

CYP26A1 Antibody (Center) Blocking Peptide - Protein Information

Name CYP26A1 {ECO:0000303|PubMed:26937021, ECO:0000312|HGNC:HGNC:2603}

Function
A cytochrome P450 monooxygenase involved in the metabolism of retinoates (RAs), the active metabolites of vitamin A, and critical signaling molecules in animals (PubMed:22020119, PubMed:9228017, PubMed:9716180). RAs exist as at least four different isomers: all- trans-RA (atRA), 9-cis-RA, 13-cis-RA, and 9,13-dicis-RA, where atRA is considered to be the biologically active isomer, although 9-cis-RA and 13-cis-RA also have activity (Probable). Catalyzes the hydroxylation of atRA primarily at C-4 and C-18, thereby contributing to the regulation of atRA homeostasis and signaling (PubMed:<a

href="http://www.uniprot.org/citations/22020119" target="_blank">22020119, PubMed:9228017, PubMed:9716180). Hydroxylation of atRA limits its biological activity and initiates a degradative process leading to its eventual elimination (Probable). Involved in the convertion of atRA to all-trans-4-oxo-RA. Able to metabolize other RAs such as 9-cis, 13-cis and 9,13-di-cis RA (By similarity) (PubMed:9228017). Can oxidize all-trans-13,14- dihydroretinoate (DRA) to metabolites which could include all-trans-4- oxo-DRA, all-trans-4-hydroxy-DRA, all-trans-5,8-epoxy-DRA, and all- trans-18-hydroxy-DRA (By similarity). May play a role in the oxidative metabolism of xenobiotics such as tazarotenic acid (PubMed:26937021).

Cellular Location
Endoplasmic reticulum membrane; Peripheral membrane protein. Microsome membrane; Peripheral membrane protein

Tissue Location
Expressed in most fetal and adult tissues with highest levels in adult liver, heart, pituitary gland, adrenal gland, placenta and regions of the brain (PubMed:9826557). Expressed at high levels in lung, pancreas, skin and uterus (at protein level) (PubMed:22020119). Lower expression level is detected in spleen, kidney, intestine and adipose tissue (at protein level) (PubMed:22020119).

CYP26A1 Antibody (Center) Blocking Peptide - Protocols

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

- Blocking Peptides

CYP26A1 Antibody (Center) Blocking Peptide - Images

CYP26A1 Antibody (Center) Blocking Peptide - Background

CYP26A1 is a member of the cytochrome P450 superfamily of enzymes. The cytochrome P450 proteins are monooxygenases which catalyze many reactions involved in drug metabolism and synthesis of cholesterol, steroids and other lipids. This endoplasmic reticulum protein acts on retinoids, including all-trans-retinoic acid (RA), with both 4-hydroxylation and 18-hydroxylation activities. This enzyme regulates the cellular level of retinoic acid which is involved in regulation of gene expression in both embryonic and adult tissues.

CYP26A1 Antibody (Center) Blocking Peptide - References

Quere,R., Blood 109 (10), 4450-4460 (2007)Lee,S.J., Pharmacogenet. Genomics 17 (3), 169-180 (2007)Heise,R., J. Invest. Dermatol. 126 (11), 2473-2480 (2006)