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AKR1C1 Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP14485b

Specification

AKR1C1 Antibody (C-term) Blocking Peptide - Product Information

Primary Accession Q04828

AKR1C1 Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 1645

Other Names
Aldo-keto reductase family 1 member C1, 111-, 20-alpha-hydroxysteroid dehydrogenase, 20-alpha-HSD, Chlordecone reductase homolog HAKRC, Dihydrodiol dehydrogenase 1/2, DD1/DD2, High-affinity hepatic bile acid-binding protein, HBAB, Indanol dehydrogenase, Trans-1, 2-dihydrobenzene-1, 2-diol dehydrogenase, AKR1C1, DDH, DDH1

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.

AKR1C1 Antibody (C-term) Blocking Peptide - Protein Information

Name AKR1C1

Synonyms DDH, DDH1

Function
Cytosolic aldo-keto reductase that catalyzes NADPH-dependent reduction of ketosteroids to hydroxysteroids. Displays broad substrate specificity with distinct positional and stereochemistry, primarily generating 20alpha-hydroxysteroids, but also 3alpha/beta- and 17beta- hydroxysteroids (PubMed:10998348, PubMed:11013348, PubMed:14672942, PubMed:19218247, PubMed:11995921, PubMed:12604236). Involved in neurosteroid metabolism. Reduces 5alpha-dihydrodeoxycorticosterone (5-alpha-DHDOC) to neuroactive steroid 3alpha,5alpha- tetrahydrodeoxycorticosterone (3alpha,5alpha-THDOC) known to alter neural excitability via allosteric activation of gamma-aminobutyric acid type A (GABAAR) receptors. Inactivates 3alpha-hydroxy-5alpha-

pregnan-20-one (3alpha,5alpha-THP) into less potent neurosteroid 3alpha,20alpha-dihydroxy-5alpha-pregnane (PubMed:11995921, PubMed:12604236). Catalyzes the reduction of progesterone to less potent progestogen (20S)-hydroxypregn-4-en-3-one likely regulating ligand availability for progesterone receptors (PubMed:10998348, PubMed:11013348, PubMed:12604236). In androgen catabolism, may predominantly act as a phase I enzyme by introducing a hydroxyl group prior to conjugation. It can nevertheless participate in the alternative phase II pathway by directly reducing sulfate- or glucuronide-conjugated androgens (PubMed:19218247). In vitro can efficiently catalyze bidirectional conversion between ketosteroids and hydroxysteroids using NADPH/NADP(+) or NADH/NAD(+) as cofactors. In vivo however, the reductase activity prevails since the major reducing cofactor NADPH inhibits NAD(+)-dependent oxidase activity (PubMed:14672942).

Cellular Location
Cytoplasm, cytosol.

Tissue Location
Expressed in all tissues tested including liver, prostate, testis, adrenal gland, brain, uterus, mammary gland and keratinocytes. Highest levels found in liver, mammary gland and brain

AKR1C1 Antibody (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

AKR1C1 Antibody (C-term) Blocking Peptide - Images

AKR1C1 Antibody (C-term) Blocking Peptide - Background

This gene encodes a member of the aldo/keto reductasesuperfamily, which consists of more than 40 known enzymes andproteins. These enzymes catalyze the conversion of aldehydes andketones to their corresponding alcohols by utilizing NADH and/orNADPH as cofactors. The enzymes display overlapping but distinctsubstrate specificity. This enzyme catalyzes the reaction ofprogesterone to the inactive form 20-alpha-hydroxy-progesterone.This gene shares high sequence identity with three other genemembers and is clustered with those three genes at chromosome10p15-p14.

AKR1C1 Antibody (C-term) Blocking Peptide - References

Joslyn, G., et al. Alcohol. Clin. Exp. Res. 34(5):800-812(2010)Wang, X., et al. PLoS ONE 5 (8), E11934 (2010) :Reding, K.W., et al. Am. J. Epidemiol. 170(10):1241-1249(2009)Chien, C.W., et al. Carcinogenesis 30(10):1813-1820(2009)Davies, N.J., et al. Cancer Res. 69(11):4769-4775(2009)