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

AKR1C4 / Chlordecone Reductase Antibody (N-Terminus)

Goat Polyclonal Antibody
Catalog # ALS12454

Specification

AKR1C4 / Chlordecone Reductase Antibody (N-Terminus) - Product Information

Application	**WB, IHC**
Primary Accession	P17516
Reactivity	**Human**
Host	**Goat**
Clonality	**Polyclonal**
Calculated MW	**37kDa KDa**

AKR1C4 / Chlordecone Reductase Antibody (N-Terminus) - Additional Information

Gene ID 1109

Other Names
Aldo-keto reductase family 1 member C4, 1.1.1.-, 3-alpha-HSD1, 3-alpha-hydroxysteroid dehydrogenase type I, 1.1.1.357, Chlordecone reductase, CDR, 1.1.1.225, Dihydrodiol dehydrogenase 4, DD-4, DD4, HAKRA, AKR1C4, CHDR

Target/Specificity
Human AKR1C4. This antibody may cross-react with AKR1C1

Reconstitution & Storage
Store at -20°C. Minimize freezing and thawing.

Precautions
AKR1C4 / Chlordecone Reductase Antibody (N-Terminus) is for research use only and not for use in diagnostic or therapeutic procedures.

AKR1C4 / Chlordecone Reductase Antibody (N-Terminus) - Protein Information

Name AKR1C4

Synonyms CHDR

Function
Cytosolic aldo-keto reductase that catalyzes the NADH and NADPH-dependent reduction of ketosteroids to hydroxysteroids. Liver specific enzyme that acts as an NAD(P)(H)-dependent 3-, 17- and 20- ketosteroid reductase on the steroid nucleus and side chain (PubMed:14672942, PubMed:10998348, PubMed:7650035, PubMed:1530633, PubMed:11158055, PubMed:10634139, PubMed:<a

href="http://www.uniprot.org/citations/19218247" target="_blank">19218247). Displays the ability to catalyze both oxidation and reduction in vitro, but most probably acts as a reductase in vivo since the oxidase activity measured in vitro is inhibited by physiological concentration of NADPH (PubMed:14672942). Acts preferentially as a 3-alpha-hydroxysteroid dehydrogenase (HSD) with a subsidiary 3-beta-HSD activity (PubMed:14672942). Catalyzes efficiently the transformation of the potent androgen 5-alpha-dihydrotestosterone (5alpha-DHT or 17beta- hydroxy-5alpha-androstan-3-one) into the less active form, 5-alpha-androstan-3-alpha,17-beta-diol (3-alpha-diol) (PubMed:11158055, PubMed:10998348, PubMed:14672942). Catalyzes the reduction of estrone into 17beta-estradiol but with low efficiency (PubMed:14672942). Metabolizes a broad spectrum of natural and synthetic therapeutic steroid and plays an important role in metabolism of androgens, estrogens, progestereone and conjugated steroids (PubMed:10998348, PubMed:14672942, PubMed:19218247). Catalyzes the biotransformation of the pesticide chlordecone (kepone) to its corresponding alcohol leading to increased biliary excretion of the pesticide and concomitant reduction of its neurotoxicity since bile is the major excretory route (PubMed:2427522).

Cellular Location
Cytoplasm, cytosol {ECO:0000250|UniProtKB:Q04828}

Tissue Location
Liver specific.

AKR1C4 / Chlordecone Reductase Antibody (N-Terminus) - Protocols

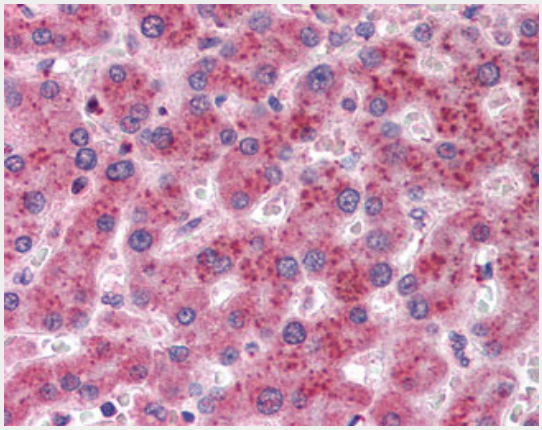
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

AKR1C4 / Chlordecone Reductase Antibody (N-Terminus) - Images

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

Antibody (0.1 ug/ml) staining of human liver lysate (35 ug protein in RIPA buffer).



Anti-AKR1C4 antibody IHC of human liver.

AKR1C4 / Chlordecone Reductase Antibody (N-Terminus) - Background

Catalyzes the transformation of the potent androgen dihydrotestosterone (DHT) into the less active form, 5-alpha- androstan-3-alpha,17-beta-diol (3-alpha-diol). Also has some 20-alpha-hydroxysteroid dehydrogenase activity. The biotransformation of the pesticide chlordecone (kepone) to its corresponding alcohol leads to increased biliary excretion of the pesticide and concomitant reduction of its neurotoxicity since bile is the major excretory route.

AKR1C4 / Chlordecone Reductase Antibody (N-Terminus) - References

Qin K.-N.,et al.J. Steroid Biochem. Mol. Biol. 46:673-679(1993).
Khanna M.,et al.J. Biol. Chem. 270:20162-20168(1995).
Khanna M.,et al.J. Steroid Biochem. Mol. Biol. 53:41-46(1995).
Kume T.,et al.Pharmacogenetics 9:763-771(1999).
Nishizawa M.,et al.Genes Cells 5:111-125(2000).