

AKR1C3 Recombinant Rabbit mAb

Catalog # AP100157

Product Information

Application	WB, IP
Primary Accession	P42330
Other Accession	P42330
Host	Rabbit
Clonality	Recombinant
Calculated MW	36853
Physical State	Liquid
Immunogen	A synthesized peptide derived from human AKR1C3
Epitope Specificity	200-250/323
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	10mM phosphate buffered saline(pH 7.4) with 150mM sodium chloride, 0.05% BSA, 0.02% Proclin300 and 50% glycerol.
SIMILARITY	Belongs to the aldo/keto reductase family.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	Cytosolic aldo-keto reductase that catalyzes the NADH and NADPH-dependent reduction of ketosteroids to hydroxysteroids. Acts as a NAD(P)(H)-dependent 3-, 17- and 20-ketosteroid reductase on the steroid nucleus and side chain and regulates the metabolism of androgens, estrogens and progesterone.

Additional Information

Gene ID	8644
Other Names	DD3; DDX; HA1753; HAKRB; HAKRe; HSD17B5; PGFS; hluPGFS; AK1C3_HUMAN; AKR1C3; 17-beta-hydroxysteroid dehydrogenase type 5 (17-beta-HSD 5); 3-alpha-HSD type II, brain; 3-alpha-hydroxysteroid dehydrogenase type 2 (3-alpha-HSD type 2); Chlordecone reductase homolog HAKRb; Dihydrodiol dehydrogenase 3 (DD-3 DD3); Dihydrodiol dehydrogenase type I; Prostaglandin F synthase (PGFS); Testosterone 17-beta-dehydrogenase 5; 1.1.1.-; 1.1.1.210; 1.1.1.53; 1.1.1.62; DDH1; KIAA0119; aldo-keto reductase family 1 member C3; hydroxysteroid (17-beta) dehydrogenase 5; aldo-keto reductase family 1, member C3 (3-alpha hydroxysteroid dehydrogenase, type II); dihydrodiol dehydrogenase X; prostaglandin F synthase; 3-alpha hydroxysteroid dehydrogenase, type II
Dilution	WB=1:500-2000, ICC/IF=1:50-200, IP=1:20-50, Flow-Cyt=1:50-100
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	AKR1C3
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 17beta-hydroxysteroids, but also 3alpha- and 20alpha- hydroxysteroids (PubMed:10998348, PubMed:11165022, PubMed:20036328, PubMed:9415401, PubMed:9927279, PubMed:10998348, PubMed:9927279). Produces potent androgens via classical and 'backdoor'/alternative pathways. In the classical androgen metabolic pathway (biosynthesis of 5alpha-dihydrotestosterone (5alpha-DHT) via testosterone), catalyzes the reduction of delta4-androstenedione to form testosterone (PubMed:10998348, PubMed:11165022, PubMed:20036328, PubMed:9415401, PubMed:9927279). In the 'backdoor' androgen metabolic pathway (biosynthesis of 5alpha-dihydrotestosterone (5alpha-DHT) via pregnanes), reduces androsterone to 5alpha-androstane-3alpha,17beta- diol preceding 5alpha-DHT secretion (PubMed:10557352, PubMed:10998348, PubMed:9415401). Reduces 5alpha-DHT to less potent androgen 5alpha-androstane-3alpha,17beta-diol, likely regulating ligand availability for androgen receptors (PubMed:10557352, PubMed:10998348, PubMed:11165022, PubMed:14672942, PubMed:7650035, PubMed:9415401). May contribute to the metabolism of adrenal-derived androgen precursors. Reduces 11-keto-4-androstene-3,17-dione (11KA4) and 11-keto-5alpha-androstane-3,17-dione (11K-Adione) into potent androgens 11-ketotestosterone (11KT) and 11-ketodihydrotestosterone (11KDHT), respectively (PubMed:31926269). In estrogen metabolism, catalyzes the conversion of estrone to potent estrogen 17beta-estradiol (PubMed:10998348, PubMed:11165022, PubMed:20036328). Acts as a prostaglandin (PG) F2alpha synthase. Displays 11-ketoreductase and 9,11-endoperoxide reductase activities and reduces PGD2 to 11beta-PGF2alpha and PGH2 to PGF2alpha (PubMed:10622721, PubMed:11165022, PubMed:15047184, PubMed:19010934, PubMed:20036328, PubMed:7650035, PubMed:9415401, PubMed:9927279). Also displays retinaldehyde reductase activity toward 9-cis-retinal (PubMed:21851338). 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:11165022, PubMed:14672942). In addition, it is able to reduce in vitro various carbonyl compounds like menadione, phenanthrenequinone and nitrobenzaldehyde (By similarity).
Cellular Location	Cytoplasm.
Tissue Location	Expressed in many tissues including adrenal gland, brain, kidney, liver, lung, mammary gland, placenta, small intestine, colon, spleen, prostate and testis. High expression in prostate and mammary gland. In the prostate, higher levels in epithelial cells than in stromal cells. In the brain, expressed in medulla, spinal cord, frontotemporal lobes, thalamus, subthalamic nuclei and amygdala. Weaker expression in the hippocampus, substantia nigra and caudate

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.