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Recombinant Anti-AKR1C3 Rabbit mAb

Catalog # AP95202

Product Information

Application	WB, FC, IP, IF/IC
Primary Accession	P42330
Reactivity	Human
Host	Rabbit
Clonality	Monoclonal
Calculated MW	36853

Additional Information

Gene ID	8644
Other Names	DDH1; HSD17B5; KIAA0119; PGFS; Aldo-keto reductase family 1 member C3; 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; HA1753; Indanol dehydrogenase; Prostaglandin F synthase; PGFS; Testosterone 17-beta-dehydrogenase 5; Trans-1, 2-dihydrobenzene-1, 2-diol dehydrogenase
Target/Specificity	KLH-conjugated synthetic peptide encompassing a sequence within human AKR1C3 protein. The exact sequence is proprietary.
Dilution	WB~~WB (1/500 - 1/1000) FC~~1:10~50 IP~~N/A IF/IC~~N/A
Format	Liquid in PBS, pH 7.3, 50% glycerol, 0.05% BSA, and 0.05% Proclin300.
Storage	Store at -20 °C.Stable for 12 months from date of receipt

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).

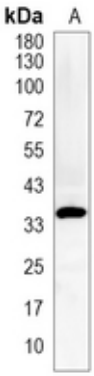
Cellular Location

Cytoplasm.

Tissue Location

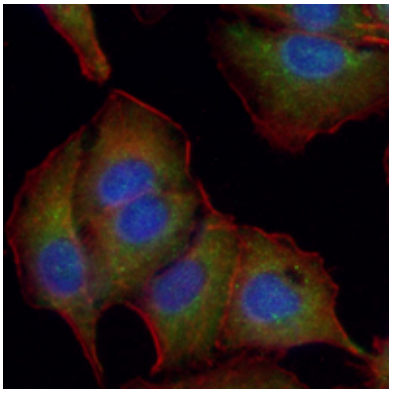
Expressed in many tissues including adrenal gland, brain, kidney, liver, lung, mammary gland, placenta, small intestine, colon, spleen, prostate and testis. High expression in prostate and mammary gland. In the prostate, higher levels in epithelial cells than in stromal cells. In the brain, expressed in medulla, spinal cord, frontotemporal lobes, thalamus, subthalamic nuclei and amygdala. Weaker expression in the hippocampus, substantia nigra and caudate

Images



Western blot analysis of AKR1C3 expression in A549 (A) whole cell lysates. (Predicted band size: 37 kD; Observed band size: 37 kD)

Immunofluorescent analysis of AKR1C3 staining in A549 cells. Formalin-fixed cells were permeabilized with 0.1% Triton X-100 in TBS for 5-10 minutes and blocked with 3% BSA-PBS for 30 minutes at room temperature. Cells were



probed with the primary antibody in 3% BSA-PBS and incubated overnight at 4 °C in a hidified chamber. Cells were washed with PBST and incubated with an AF488-conjugated secondary antibody (green) in PBS at room temperature in the dark. Phalloidin - AF594 was used to stain Actin filaments (red). DAPI was used to stain the cell nuclei (blue).

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