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AKR1C3 Antibody (N-term)

Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP10158A

Product Information

Application	WB, FC, E
Primary Accession	P42330
Other Accession	NP_003730.4
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB22754
Calculated MW	36853
Antigen Region	10-36

Additional Information

Gene ID	8644
Other Names	Aldo-keto reductase family 1 member C3, 1---, 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, AKR1C3, DDH1, HSD17B5, KIAA0119, PGFS
Target/Specificity	This AKR1C3 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 10-36 amino acids from the N-terminal region of human AKR1C3.
Dilution	WB~~1:1000 FC~~1:10~50 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	AKR1C3 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

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

Background

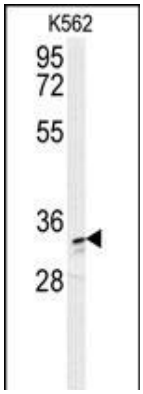
This gene encodes a member of the aldo/keto reductase superfamily, which consists of more than 40 known enzymes and proteins. These enzymes catalyze the conversion of aldehydes and ketones to their corresponding alcohols by utilizing NADH and/or NADPH as cofactors. The enzymes display overlapping but distinct substrate specificity. This enzyme catalyzes the reduction of prostaglandin (PG) D2, PGH2 and phenanthrenequinone (PQ), and the oxidation of 9alpha,11beta-PGF2 to PGD2. It may play an important role in the pathogenesis of allergic diseases such as asthma, and may also have a role in controlling cell growth

and/or differentiation. This gene shares high sequence identity with three other gene members and is clustered with those three genes at chromosome 10p15-p14.

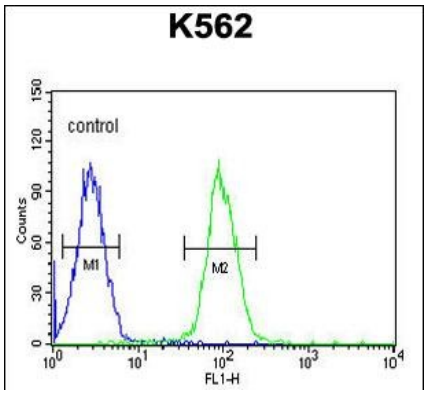
References

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Liu, C.Y., et al. Carcinogenesis 31(7):1259-1263(2010)
Rose, J.E., et al. Mol. Med. 16 (7-8), 247-253 (2010) :
Wang, X., et al. PLoS ONE 5 (8), E11934 (2010) :
Zakharov, V., et al. Int J Clin Exp Pathol 3(6):608-617(2010)

Images



AKR1C3 Antibody (N-term) (Cat. #AP10158a) western blot analysis in K562 cell line lysates (35ug/lane).This demonstrates the AKR1C3 antibody detected the AKR1C3 protein (arrow).



AKR1C3 Antibody (N-term) (Cat. #AP10158a) flow cytometric analysis of K562 cells (right histogram) compared to a negative control cell (left histogram).FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

Citations

- Induction of PGF2α synthesis by cortisol through GR dependent induction of CBR1 in human amnion fibroblasts.

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