

## AKR1C3 Antibody (N-term) Blocking peptide

Synthetic peptide Catalog # BP10158a

### **Specification**

## AKR1C3 Antibody (N-term) Blocking peptide - Product Information

Primary Accession P42330
Other Accession NP\_003730.4

## AKR1C3 Antibody (N-term) Blocking peptide - 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

#### **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.

## AKR1C3 Antibody (N-term) Blocking peptide - Protein Information

## Name AKR1C3

#### **Function**

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 (PubMed:<a href="http://www.uniprot.org/citations/10622721" target="\_blank">10622721</a>, PubMed:<a href="http://www.uniprot.org/citations/11165022" target="\_blank">11165022</a>, PubMed:<a href="http://www.uniprot.org/citations/7650035" target="\_blank">7650035</a>, PubMed:<a href="http://www.uniprot.org/citations/9415401" target="\_blank">9415401</a><a href="http://www.uniprot.org/citations/9927279" target="\_blank">9927279</a>, PubMed:<a href="http://www.uniprot.org/citations/9927279" target="\_blank">9927279</a>). 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:<a href="http://www.uniprot.org/citations/14672942" target="\_blank">14672942</a>, PubMed:<a href="http://www.uniprot.org/citations/14672942" target="\_blank">14672942</a>, PubMed:<a



href="http://www.uniprot.org/citations/11165022" target=" blank">11165022</a>). Acts preferentially as a 17-ketosteroid reductase and has the highest catalytic efficiency of the AKR1C enzyme for the reduction of delta4-androstenedione to form testosterone (PubMed:<a href="http://www.uniprot.org/citations/20036328" target="\_blank">20036328</a>). Reduces prostaglandin (PG) D2 to 11beta-prostaglandin F2, progesterone to 20alpha-hydroxyprogesterone and estrone to 17beta-estradiol (PubMed:<a href="http://www.uniprot.org/citations/15047184" target=" blank">15047184</a>, PubMed:<a href="http://www.uniprot.org/citations/20036328" target="blank">20036328</a>, PubMed:<a href="http://www.uniprot.org/citations/10622721" target="blank">10622721</a>, PubMed:<a href="http://www.uniprot.org/citations/11165022" target="\_blank">11165022</a>, PubMed:<a href="http://www.uniprot.org/citations/10998348" target="blank">10998348</a>, PubMed:<a href="http://www.uniprot.org/citations/19010934" target="blank">19010934</a>). 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) (PubMed: <a href="http://www.uniprot.org/citations/10998348" target=" blank">10998348</a>, PubMed:<a href="http://www.uniprot.org/citations/14672942" target="blank">14672942</a>, PubMed:<a href="http://www.uniprot.org/citations/11165022" target="blank">11165022</a>, PubMed:<a href="http://www.uniprot.org/citations/7650035" target="\_blank">7650035</a>, PubMed:<a href="http://www.uniprot.org/citations/9415401" target=" blank">9415401</a>, PubMed:<a href="http://www.uniprot.org/citations/10557352" target=" blank">10557352</a>). Also displays retinaldehyde reductase activity toward 9-cis-retinal (PubMed: <a href="http://www.uniprot.org/citations/21851338" target=" blank">21851338</a>).

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

## AKR1C3 Antibody (N-term) Blocking peptide - Protocols

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

### Blocking Peptides

AKR1C3 Antibody (N-term) Blocking peptide - Images

# AKR1C3 Antibody (N-term) Blocking peptide - Background

This gene encodes a member of the aldo/keto reductasesuperfamily, 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/orNADPH 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/ordifferentiation. This gene shares high sequence identity with three other gene members and is clustered with those three genes atchromosome 10p15-p14.

#### AKR1C3 Antibody (N-term) Blocking peptide - References

Canzian, F., et al. Hum. Mol. Genet. 19(19):3873-3884(2010)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)