

## **HSD11B2** Polyclonal Antibody

Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP57774

## **Product Information**

Application WB, IHC-P, IF
Primary Accession P80365
Reactivity Rat, Dog
Host Rabbit
Clonality Polyclonal
Calculated MW 44127
Physical State Liquid

Immunogen KLH conjugated synthetic peptide derived from human HSD11B2

**Epitope Specificity** 151-250/405

**Isotype** IgG

**Purity** affinity purified by Protein A

**Buffer** Preservative: 0.02% Proclin300, Constituents: 1% BSA, 0.01M PBS, pH7.4.

**SUBCELLULAR LOCATION** Microsome. Endoplasmic reticulum.

**SIMILARITY** Belongs to the short-chain dehydrogenases/reductases (SDR) family. **SUBUNIT** Interacts with ligand-free cytoplasmic NR3C2.

**DISEASE**Defects in HSD11B2 are the cause of apparent mineralocorticoid excess (AME)

[MIM:218030]. An autosomal recessive form of low-renin hypertension. It is usually diagnosed within the first years of life and is characterized by polyuria and polydipsia, failure to thrive, hypernatremia, severe hypertension with low renin and aldosterone levels, profound hypokalemia with metabolic alkalosis,

and most often nephrocalcinosis.

**Important Note** This product as supplied is intended for research use only, not for use in

human, therapeutic or diagnostic applications.

**Background Descriptions** There are at least two isozymes of the corticosteroid 11-beta-dehydrogenase,

a microsomal enzyme complex responsible for the interconversion of cortisol and cortisone. The type I isozyme has both 11-beta-dehydrogenase (cortisol to cortisone) and 11-oxoreductase (cortisone to cortisol) activities. The type II isozyme, encoded by this gene, has only 11-beta-dehydrogenase activity. In aldosterone-selective epithelial tissues such as the kidney, the type II isozyme catalyzes the glucocorticoid cortisol to the inactive metabolite cortisone, thus preventing illicit activation of the mineralocorticoid receptor. In tissues that do not express the mineralocorticoid receptor, such as the placenta and testis, it protects cells from the growth-inhibiting and/or pro-apoptotic effects of cortisol, particularly during embryonic development. Mutations in this gene cause the syndrome of apparent mineralocorticoid excess and hypertension.

[provided by RefSeq, Feb 2010]

## **Additional Information**

**Gene ID** 3291

Other Names Corticosteroid 11-beta-dehydrogenase isozyme 2, 1.1.1.-,

11-beta-hydroxysteroid dehydrogenase type 2, 11-DH2, 11-beta-HSD2, 11-beta-hydroxysteroid dehydrogenase type II, 11-HSD type II, 11-beta-HSD type II, NAD-dependent 11-beta-hydroxysteroid dehydrogenase, 11-beta-HSD,

Short chain dehydrogenase/reductase family 9C member 3, HSD11B2

(HGNC:5209)

**Target/Specificity** Found in placenta, kidney, pancreas, prostate, ovary, small intestine and

colon.

**Dilution** WB=1:500-2000,IHC-P=1:100-500,IF=1:100-500

Format 0.01M TBS(pH7.4) with 1% BSA, 0.09% (W/V) sodium azide and 50% Glyce

**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 HSD11B2 ( HGNC:5209)

**Function** Catalyzes the conversion of biologically active 11beta-

hydroxyglucocorticoids (11beta-hydroxysteroid) such as cortisol, to inactive 11-ketoglucocorticoids (11-oxosteroid) such as cortisone, in the presence of

NAD(+) (PubMed: 10497248, PubMed: 12788846, PubMed: 17314322,

PubMed:22796344, PubMed:27927697, PubMed:30902677,

PubMed:<u>33387577</u>, PubMed:<u>7859916</u>, PubMed:<u>8538347</u>). Functions as a dehydrogenase (oxidase), thereby decreasing the concentration of active glucocorticoids, thus protecting the nonselective mineralocorticoid receptor from occupation by glucocorticoids (PubMed:<u>10497248</u>, PubMed:<u>12788846</u>, PubMed:<u>17314322</u>, PubMed:<u>33387577</u>, PubMed:<u>7859916</u>). Plays an important

PubMed:<u>17314322</u>, PubMed:<u>33387577</u>, PubMed:<u>7859916</u>). Plays an imporrole in maintaining glucocorticoids balance during preimplantation and protects the fetus from excessive maternal corticosterone exposure (By similarity). Catalyzes the oxidation of 11beta-hydroxytestosterone (11beta,17beta-dihydroxyandrost-4-ene-3-one) to 11-ketotestosterone (17beta-hydroxyandrost-4-ene-3,11-dione), a major bioactive androgen (PubMed:<u>22796344</u>, PubMed:<u>27927697</u>). Catalyzes the conversion of

11beta-hydroxyandrostenedione (11beta-hydroxyandrost- 4-ene-3,17-dione) to 11-ketoandrostenedione (androst-4-ene-3,11,17- trione), which can be further metabolized to 11-ketotestosterone (PubMed:27927697). Converts 7-beta-25-dihydroxycholesterol to 7-oxo-25- hydroxycholesterol in vitro

(PubMed:30902677). 7-beta-25- dihydroxycholesterol (not

7-oxo-25-hydroxycholesterol) acts as a ligand for the G-protein-coupled receptor (GPCR) Epstein-Barr virus-induced gene 2 (EBI2) and may thereby regulate immune cell migration (PubMed:30902677). May protect ovulating oocytes and fertilizing spermatozoa from the adverse effects of cortisol (By

similarity).

**Cellular Location** Microsome. Endoplasmic reticulum

**Tissue Location** Expressed in kidney, placenta, pancreas, prostate, ovary, small intestine and

colon, and in lower levels in the spleen and testis (PubMed:7859916). At midgestation, expressed at high levels in placenta and in fetal kidney and, at

much lower levels, in fetal lung and testis (PubMed:8530071).