

HSD11B2 Antibody (Center)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP9764c

Specification

HSD11B2 Antibody (Center) - Product Information

Application	WB, IHC-P, FC,E
Primary Accession	P80365
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit Ig
Calculated MW	44127
Antigen Region	277-306

HSD11B2 Antibody (Center) - Additional Information

Gene ID 3291

Other Names

Corticosteroid 11-beta-dehydrogenase isozyme 2, 11-, 11-beta-hydroxysteroid dehydrogenase type 2, 11-DH2, 11-beta-HSD2, 11-beta-hydroxysteroid dehydrogenase type II, -HSD11 type II, NAD-dependent 11-beta-hydroxysteroid dehydrogenase, 11-beta-HSD, HSD11B2, HSD11K

Target/Specificity

This HSD11B2 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 277-306 amino acids from the Central region of human HSD11B2.

Dilution

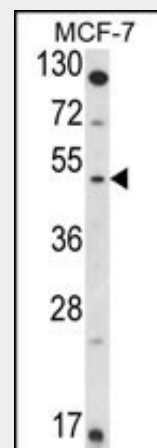
WB~~1:1000
IHC-P~~1:50~100
FC~~1:10~50

Format

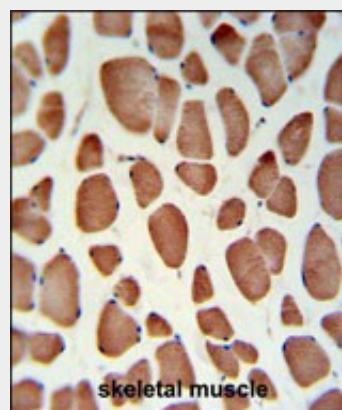
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2



Western blot analysis of HSD11B2 Antibody (Center) (Cat. #AP9764c) in MCF-7 cell line lysates (35ug/lane). HSD11B2 (arrow) was detected using the purified Pab.



HSD11B2 Antibody (Center) (Cat. #AP9764c) IHC analysis in formalin fixed and paraffin embedded skeletal muscle followed by peroxidase conjugation of the secondary antibody and DAB staining. This data demonstrates the use of the HSD11B2 Antibody (Center) for immunohistochemistry. Clinical relevance has not been evaluated.

weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

HSD11B2 Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

HSD11B2 Antibody (Center) - Protein Information

Name HSD11B2 ([HGNC:5209](#))

Function

Catalyzes the conversion of cortisol to the inactive metabolite cortisone. Modulates intracellular glucocorticoid levels, thus protecting the nonselective mineralocorticoid receptor from occupation by glucocorticoids.

Cellular Location

Microsome. Endoplasmic reticulum

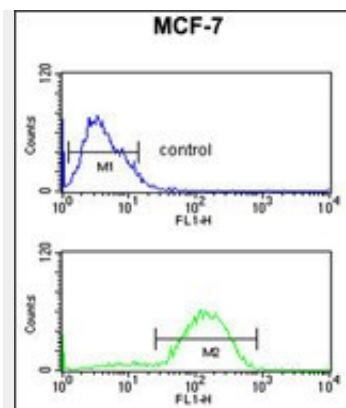
Tissue Location

Expressed in kidney, pancreas, prostate, ovary, small intestine and colon. At midgestation, expressed at high levels in placenta and in fetal kidney and, at much lower levels, in fetal lung and testis (PubMed:8530071).

HSD11B2 Antibody (Center) - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)



HSD11B2 Antibody (Center) (Cat. #AP9764c) flow cytometric analysis of MCF-7 cells (bottom histogram) compared to a negative control cell (top histogram). FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

HSD11B2 Antibody (Center) - Background

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.

HSD11B2 Antibody (Center) - References

- Li, J., et al. Breast Cancer Res. 12 (2), R19 (2010)
Ni, X.T., et al. Placenta 30(12):1023-1028(2009)
Mericq, V., et al. Eur. J. Endocrinol. 161(3):419-425(2009)
Stark, M.J., et al. Am. J. Physiol. Regul. Integr.

Comp. Physiol. 297 (2), R510-R514 (2009)
Lepenes, J., et al. Clin. Exp. Hypertens.
31(4):376-379(2009)