abcam

Product datasheet

Recombinant Human C11B2/CYP11B2 protein (His tag) ab224858

1 Image

Description

Product name Recombinant Human C11B2/CYP11B2 protein (His tag)

Purity > 90 % SDS-PAGE.

Expression system Escherichia coli

Accession P19099

Protein length Full length protein

Animal free No

Nature Recombinant

Species Human

Sequence GTRAARAPRTVLPFEAMPQHPGNRWLRLLQWREQGYEH

LHLEMHQTFQE

LGPIFRYNLGGPRMVCVMLPEDVEKLQQVDSLHPCRMILE

PWVAYRQHRG

HKCGVFLLNGPEWRFNRLRLNPDVLSPKAVQRFLPMVD

AVARDFSQALKK

KVLQNARGSLTLDVQPSIFHYTIEASNLALFGERLGLVGHS

PSSASLNFL

 ${\sf HALEVMFKSTVQLMFMPRSLSRWISPKVWKEHFEAWDCI}$

FQYGDNCIQKI

YQELAFNRPQHYTGIVAELLLKAELSLEAIKANSMELTAGS

VDTTAFPLL

MTLFELARNPDVQQILRQESLAAAASISEHPQKATTELPLL

RAALKETLR

LYPVGLFLERVVSSDLVLQNYHIPAGTLVQVFLYSLGRNAA

LFPRPERYN

PQRWLDIRGSGRNFHHVPFGFGMRQCLGRRLAEAEMLLL LHHVLKHFLVE TLTQEDIKMVYSFILRPGTSPLLTFRAIN

Predicted molecular weight 71 kDa including tags

Amino acids 25 to 503

Tags His tag N-Terminus

Additional sequence information This product is the full-length mature protein from aa 25-503 with 6xHis-SUMO tag at the N-

1

Specifications

Our **Abpromise guarantee** covers the use of **ab224858** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications SDS-PAGE

Form Liquid

Additional notes This product was previously labelled as C11B2

Preparation and Storage

Stability and Storage Shipped at 4°C. Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.

pH: 7.2

Constituents: 50% Glycerol (glycerin, glycerine), Tris buffer

General Info

Function Preferentially catalyzes the conversion of 11-deoxycorticosterone to aldosterone via

corticosterone and 18-hydroxycorticosterone.

Involvement in disease Defects in CYP11B2 are the cause of corticosterone methyloxidase type 1 deficiency (CMO-1

> deficiency) [MIM:203400]; also known as aldosterone deficiency due to defect in 18-hydroxylase or aldosterone deficiency I. CMO-1 deficiency is an autosomal recessive disorder of aldosterone biosynthesis. There are two biochemically different forms of selective aldosterone deficiency be termed corticosterone methyloxidase (CMO) deficiency type 1 and type 2. In CMO-1 deficiency, aldosterone is undetectable in plasma, while its immediate precursor, 18-hydroxycorticosterone,

is low or normal.

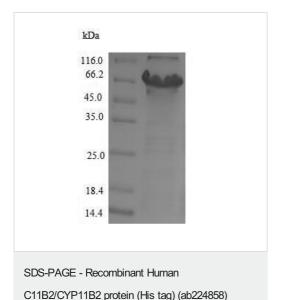
Defects in CYP11B2 are the cause of corticosterone methyloxidase type 2 deficiency (CMO-2 deficiency) [MIM:610600]. CMO-2 is an autosomal recessive disorder of aldosterone biosynthesis. In CMO-2 deficiency, aldosterone can be low or normal, but at the expense of increased secretion of 18-hydroxycorticosterone. Consequently, patients have a greatly increased ratio of 18-hydroxycorticosterone to aldosterone and a low ratio of corticosterone to 18hydroxycorticosterone in serum.

Defects in CYP11B2 are a cause of familial hyperaldosteronism type 1 (FH1) [MIM:103900]. It is a disorder characterized by hypertension, variable hyperaldosteronism, and abnormal adrenal steroid production, including 18-oxocortisol and 18-hydroxycortisol. There is significant phenotypic heterogeneity, and some individuals never develop hypertension. Note=The molecular defect causing hyperaldosteronism familial type 1 is an anti-Lepore-type fusion of the CYP11B1 and CYP11B2 genes. The hybrid gene has the promoting part of CYP11B1, ACTH-sensitive, and the coding part of CYP11B2.

Sequence similarities Belongs to the cytochrome P450 family.

Cellular localization Mitochondrion membrane.

Images



(Tris-Glycine gel) Discontinuous SDS-PAGE (reduced) analysis of ab224858 with 5% enrichment gel and 15% separation gel.

Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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