SunLong Biotech Co.,LTD
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[Primary Antibody]

Rabbit Anti-CYP11B2 antibody

Catalog Number: SL23822R

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	CYP11B2
Immunogen:	KLH conjugated synthetic peptide derived from human CYP11B2 : 221-320/503
Purification:	affinity purified by Protein A
Concentration:	1mg/1ml
Storage:	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4℃. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.
Applications:	WB=1:500-2000 （Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Human
Predicted MW.:	55kDa
Subcellular Location:	cytoplasmic The cell membrane
Background:	This gene encodes a member of the cytochrome P450 superfamily of enzymes. The cytochrome P450 proteins are monooxygenases which catalyze many reactions involved in drug metabolism and synthesis of cholesterol, steroids and other lipids. This protein localizes to the mitochondrial inner membrane. The enzyme has steroid 18-hydroxylase activity to synthesize aldosterone and 18-oxocortisol as well as steroid 11 beta-hydroxylase activity. Mutations in this gene cause corticosterone methyl oxidase deficiency. [provided by RefSeq, Jul 2008]. **Function:** Preferentially catalyzes the conversion of 11-deoxycorticosterone to aldosterone via corticosterone and 18-hydroxycorticosterone. **Subcellular Location:** Mitochondrion membrane. 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

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	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 18-hydroxycorticosterone 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. **Similarity:** Belongs to the cytochrome P450 family. **Database links:** **Entrez Gene:** 1585 Human **Entrez Gene:** 13072 Mouse **Entrez Gene:** 24294 Rat Omim: 124080 Human **SwissProt:** P19099 Human **SwissProt:** P15539 Mouse **SwissProt:** P30099 Rat **Unigene:** 632054 Human **Unigene:** 377079 Mouse **Unigene:** 144549 Rat

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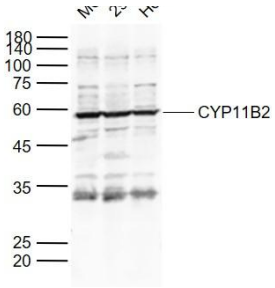
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[VALIDATION IMAGES]



Sample:
Lane 1: MCF-7 (Human) Cell Lysate at 30 ug
Lane 2: 293T (Human) Cell Lysate at 30 ug
Lane 3: HepG2 (Human) Cell Lysate at 30 ug
Primary: Anti-CYP11B2 (SL23822R) at 1/1000 dilution
Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution
Predicted band size: 55 kD
Observed band size: 58 kD

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