

## C11B2 Rabbit pAb

CatalogNo: YN1729

### | Key Features

#### Host Species

- Rabbit

#### Reactivity

- Human, Mouse, Rat

#### Applications

- WB, ELISA

#### MW

- 55kD (Observed)

#### Isotype

- IgG

### | Recommended Dilution Ratios

**WB 1:500-2000**

**ELISA 1:5000-20000**

### | Storage

**Storage\*** -15°C to -25°C/1 year (Do not lower than -25°C)

**Formulation** Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

### | Basic Information

**Clonality** Polyclonal

### | Immunogen Information

**Immunogen** Synthesized peptide derived from human protein . at AA range: 280-360

**Specificity** C11B2 Polyclonal Antibody detects endogenous levels of protein.

### | Target Information

**Gene name** CYP11B2

**Protein Name** Cytochrome P450 11B2, mitochondrial (Aldosterone synthase) (ALDOS) (Aldosterone-synthesizing enzyme) (CYPXIB2) (Cytochrome P-450Aldo) (Cytochrome P-450C18) (Steroid 18-hydroxylase)

Organism	Gene ID	UniProt ID
Human	<a href="#">1585</a> ;	<a href="#">P19099</a> ;
Mouse		<a href="#">P15539</a> ;
Rat		<a href="#">P30099</a> ;

**Cellular Localization** Mitochondrion inner membrane ; Peripheral membrane protein .

**Tissue specificity** Adrenal gland,Blood,

**Function** Catalytic activity:A steroid + reduced adrenal ferredoxin + O(2) = an 11-beta-hydroxysteroid + oxidized adrenal ferredoxin + H(2)O.,Catalytic activity:Corticosterone + reduced adrenal ferredoxin + O(2) = 18-hydroxycorticosterone + oxidized adrenal ferredoxin + H(2)O.,cofactor:Heme group.,Disease:An anti-Lepore-type fusion of the CYP11B2 and CYP11B1 genes is a cause of glucocorticoid-remediable aldosteronism (GRA) [MIM:103900].,Disease:Defects in CYP11B2 are the cause of corticosterone methyloxidase type 1 deficiency (CMO-1 deficiency) [MIM:203400]; also called 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.,Disease: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.,Function:Preferentially catalyzes the conversion of 11-deoxycorticosterone to aldosterone via corticosterone and 18-hydroxycorticosterone.,online information:CYP11B2 entry,similarity:Belongs to the cytochrome P450 family.,

## Validation Data

## Contact information

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**C11B2 Rabbit pAb**

