

**CYP11A1 Antibody**

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**For Research Use Only. Not for Use in Diagnostic Procedures.**

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP	H M R	Endogenous	50	Rabbit	#P05108	1583

**Product Usage Information****Application**

Western Blotting  
Immunoprecipitation

**Dilution**

1:1000  
1:50

**Storage**

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at -20°C. Do not aliquot the antibody.

**Specificity/Sensitivity**

CYP11A1 Antibody recognizes endogenous levels of total CYP11A1 protein.

**Source / Purification**

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues near the amino terminus of human CYP11A1 protein. Antibodies are purified by protein A and peptide affinity chromatography.

**Background**

In steroidogenic tissues, such as the adrenal cortex, testis, ovary, and placenta, all steroids are synthesized from the common precursor cholesterol. Two families of steroidogenic enzymes, cytochrome P450 hydroxylase enzymes (CYP) and hydroxysteroid dehydrogenases (HSD), catalyze the production of most steroids. There are six distinct steroid hydroxylases, which are cytochrome P450 enzymes encoded by the steroidogenic *CYP* gene family (1). The cytochrome P450<sub>scc</sub> (cholesterol side-chain cleavage enzyme) encoded by *CYP11A1* catalyzes the first and rate-limiting step in steroidogenesis, conversion of cholesterol into pregnenolone (2).

CYP11A1, located in the inner membrane of mitochondria, cooperates with two coenzymes, ferredoxin and ferredoxin reductase, to carry out three successive oxidation-reduction reactions of cholesterol (3-5). In the adrenal cortex, testis, and ovary, CYP11A1 expression is regulated by the cAMP-PKA pathway (6), and the transcription factor SF1/NR5A1 has been shown to play a central role in mediating the cAMP signal on the *CYP11A1* promoter within steroidogenic cells of the adrenal cortex and gonads (7). Defects in CYP11A1 are the cause of adrenal insufficiency congenital with 46, XY sex reversal (AICSR), which is a rare disorder that can present as acute adrenal insufficiency in infancy or childhood (8,9).

**Background References**

1. Nelson, D.R. et al. (1993) *DNA Cell Biol* 12, 1-51.
2. Richards, J.S. et al. (1987) *Recent Prog Horm Res* 43, 231-76.
3. Hanukoglu, I. and Jefcoate, C.R. (1980) *J Biol Chem* 255, 3057-61.
4. Hanukoglu, I. et al. (1981) *J Biol Chem* 256, 4329-35.
5. Hanukoglu, I. et al. (1981) *J Biol Chem* 256, 4321-8.
6. Hu, M.C. et al. (1991) *Biochem J* 274 ( Pt 3), 813-7.
7. Watanabe, N. et al. (1994) *Eur J Biochem* 222, 825-34.
8. Tajima, T. et al. (2001) *J Clin Endocrinol Metab* 86, 3820-5.
9. Katsumata, N. et al. (2002) *J Clin Endocrinol Metab* 87, 3808-13.

**Species Reactivity**

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

**Western Blot Buffer**

**IMPORTANT:** For western blots, incubate membrane with diluted primary antibody in 5% w/v nonfat dry milk, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

**Applications Key**

**W:** Western Blotting **IP:** Immunoprecipitation

**Cross-Reactivity Key**

**H:** Human **M:** Mouse **R:** Rat

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