

# Cytochrome P450 17A1 Rabbit pAb

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Catalog # AP52080

## Product Information

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<b>Application</b>	WB, IHC-P, IHC-F, IF
<b>Primary Accession</b>	<a href="#">P05093</a>
<b>Reactivity</b>	Mouse, Rat
<b>Predicted</b>	Human, Horse, Rabbit, Sheep
<b>Host</b>	Rabbit
<b>Clonality</b>	Polyclonal
<b>Calculated MW</b>	57371
<b>Physical State</b>	Liquid
<b>Immunogen</b>	KLH conjugated synthetic peptide derived from human P45017A1/Cytochrome P450 17A1
<b>Epitope Specificity</b>	24-65/508
<b>Isotype</b>	IgG
<b>Purity</b>	affinity purified by Protein A
<b>Buffer</b>	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
<b>SUBCELLULAR LOCATION</b>	Membrane.
<b>SIMILARITY</b>	Belongs to the cytochrome P450 family.
<b>Post-translational modifications</b>	Phosphorylation is necessary for 17,20-lyase, but not for 17-alpha-hydroxylase activity.
<b>DISEASE</b>	Defects in CYP17A1 are the cause of adrenal hyperplasia type 5 (AH5) [MIM:202110]. AH5 is a form of congenital adrenal hyperplasia, a common recessive disease due to defective synthesis of cortisol. Congenital adrenal hyperplasia is characterized by androgen excess leading to ambiguous genitalia in affected females, rapid somatic growth during childhood in both sexes with premature closure of the epiphyses and short adult stature. Four clinical types: "salt wasting" (SW, the most severe type), "simple virilizing" (SV, less severely affected patients), with normal aldosterone biosynthesis, "non-classic form" or late onset (NC or LOAH), and "cryptic" (asymptomatic).
<b>Important Note</b>	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
<b>Background Descriptions</b>	Cytochrome P450 17A1 (CYP17A1) belongs to the cytochrome P450 family; it plays a role in the conversion of pregnenolone and progesterone into their 17-alpha-hydroxylated products and subsequently to dehydroepiandrosterone (DHEA) and androstenedione. CYP17A1 also catalyzes both the 17-alpha-hydroxylation and the 17,20-lyase reaction. CYP17A1 is involved in sexual development during fetal life and at puberty. Defects in CYP17A1 are the cause of adrenal hyperplasia type 5 (AH5). AH5 is a form of congenital adrenal hyperplasia, a common recessive disease due to defective synthesis of cortisol.

## Additional Information

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<b>Gene ID</b>	1586
<b>Other Names</b>	Steroid 17-alpha-hydroxylase/17, 20 lyase, 1.14.14.19, 17-alpha-hydroxyprogesterone aldolase, 1.14.14.32, CYPXVII, Cytochrome P450 17A1, Cytochrome P450-C17, Cytochrome P450c17, Steroid 17-alpha-monooxygenase, CYP17A1 {ECO:0000303 PubMed:19793597, ECO:0000312 HGNC:HGNC:2593}
<b>Dilution</b>	WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500
<b>Storage</b>	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

## Protein Information

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<b>Name</b>	CYP17A1 {ECO:0000303 PubMed:19793597, ECO:0000312 HGNC:HGNC:2593}
<b>Function</b>	A cytochrome P450 monooxygenase involved in corticoid and androgen biosynthesis (PubMed: <a href="#">22266943</a> , PubMed: <a href="#">25301938</a> , PubMed: <a href="#">27339894</a> , PubMed: <a href="#">9452426</a> ). Catalyzes 17-alpha hydroxylation of C21 steroids, which is common for both pathways. A second oxidative step, required only for androgen synthesis, involves an acyl-carbon cleavage. The 17-alpha hydroxy intermediates, as part of adrenal glucocorticoids biosynthesis pathway, are precursors of cortisol (Probable) (PubMed: <a href="#">25301938</a> , PubMed: <a href="#">9452426</a> ). Hydroxylates steroid hormones, pregnenolone and progesterone to form 17-alpha hydroxy metabolites, followed by the cleavage of the C17-C20 bond to form C19 steroids, dehydroepiandrosterone (DHEA) and androstenedione (PubMed: <a href="#">22266943</a> , PubMed: <a href="#">25301938</a> , PubMed: <a href="#">27339894</a> , PubMed: <a href="#">36640554</a> , PubMed: <a href="#">9452426</a> ). Has 16-alpha hydroxylase activity. Catalyzes 16-alpha hydroxylation of 17-alpha hydroxy pregnenolone, followed by the cleavage of the C17-C20 bond to form 16-alpha-hydroxy DHEA (PubMed: <a href="#">36640554</a> ). Also 16-alpha hydroxylates androgens, relevant for estriol synthesis (PubMed: <a href="#">25301938</a> , PubMed: <a href="#">27339894</a> ). Mechanistically, uses molecular oxygen inserting one oxygen atom into a substrate, and reducing the second into a water molecule, with two electrons provided by NADPH via cytochrome P450 reductase (CPR; NADPH-ferrihemoprotein reductase) (PubMed: <a href="#">22266943</a> , PubMed: <a href="#">25301938</a> , PubMed: <a href="#">27339894</a> , PubMed: <a href="#">9452426</a> ).
<b>Cellular Location</b>	Endoplasmic reticulum membrane. Microsome membrane

## Background

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Cytochrome P450 17A1 (CYP17A1) belongs to the cytochrome P450 family; it plays a role in the conversion of pregnenolone and progesterone into their 17-alpha-hydroxylated products and subsequently to dehydroepiandrosterone (DHEA) and androstenedione. CYP17A1 also catalyzes both the 17-alpha-hydroxylation and the 17,20-lyase reaction. CYP17A1 is involved in sexual development during fetal life and at puberty. Defects in CYP17A1 are the cause of adrenal hyperplasia type 5 (AH5). AH5 is a form of congenital adrenal hyperplasia, a common recessive disease due to defective synthesis of cortisol.

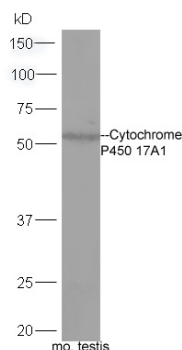
## References

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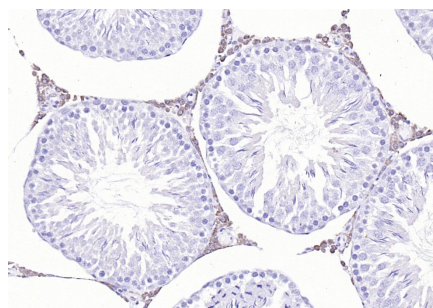
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Bradshaw K.D.,et al.Mol. Endocrinol. 1:348-354(1987).  
Brentano S.T.,et al.Mol. Endocrinol. 4:1972-1979(1990).  
Kagimoto M.,et al.Mol. Endocrinol. 2:564-570(1988).

## Images



Sample: Testis (Mouse) Lysate at 40 ug  
Primary: Anti-Cytochrome P450 17A1 (AP52080) at 1/300 dilution  
Secondary: HRP conjugated Goat-Anti-rabbit IgG (AP52080-HRP) at 1/5000 dilution  
Predicted band size: 57 kD  
Observed band size: 57 kD



Paraformaldehyde-fixed, paraffin embedded (rat testis);  
Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (Cytochrome P450 17A1) Polyclonal Antibody, Unconjugated (AP52080) at 1:200 overnight at 4°C, followed by operating according to SP Kit(Rabbit) (sp-0023) instructions and DAB staining.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.