

## Rabbit Anti-HSD3B2 antibody

SL16552R

Product Name:	HSD3B2
Chinese Name:	2型肾上腺皮质增生症蛋白抗体
Alias:	3 beta HSD adrenal and gonadal type; 3 beta HSD II; 3 beta HSD type II; 3 beta hydroxy 5 ene steroid dehydrogenase; 3 beta hydroxy Delta(5) steroid dehydrogenase; 3 beta hydroxysteroid dehydrogenase/Delta 5>4-isomerase type 2; 3 beta-hydroxysteroid dehydrogenase type II, delta 5-delta 4-isomerase type II, 3 beta-HSD type II; 3 beta- hydroxysteroid dehydrogenase/Delta 5>4-isomerase type II; 3 beta-hydroxysteroid dehydrogenase/Delta 5>4-isomerase type II; 3 beta-hydroxysteroid dehydrogenase/Delta 5>4-isomerase type 2; 3-beta-HSD II; 3-beta-hydroxy-5-ene steroid dehydrogenase; 3-beta-hydroxy-Delta(5)-steroid dehydrogenase; 3BHS2_HUMAN; ADRENAL HYPERPLASIA II; beta-hydroxysteroid dehydrogenase/Delta 5>4-isomerase type 2; delta 5 delta 4 isomerase type II; Delta-5- 3-ketosteroid isomerase; HSD3B; HSD3B2; HSDB; HSDB3B; hydroxy delta 5 steroid dehydrogenase, 3 beta and steroid delta isomerase 2; Progesterone reductase.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Dog,Pig,
Applications:	ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800ICC=1:100-500IF=1:100- 500 (Paraffin sections need antigen repair) not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	40kDa
Cellular localization:	cytoplasmic <u>Mitochondrion</u>
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthetic peptide derived from human HSD3B2:
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized

	antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. 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
PubMod.	DubMed
	3-beta-HSD is a bifunctional enzyme, that catalyzes the oxidative conversion of Delta(5)-ene-3-beta-hydroxy steroid, and the oxidative conversion of ketosteroids. The 3-beta-HSD enzymatic system plays a crucial role in the biosynthesis of all classes of hormonal steroids.
	Function: 3-beta-HSD is a bifunctional enzyme, that catalyzes the oxidative conversion of Delta(5)-ene-3-beta-hydroxy steroid, and the oxidative conversion of ketosteroids. The 3-beta-HSD enzymatic system plays a crucial role in the biosynthesis of all classes of hormonal steroids.
	Subunit: Expressed in adrenal gland, testis and ovary.
	Subcellular Location: Endoplasmic reticulum membrane. Mitochondrion membrane.
Product Detail:	<b>Tissue Specificity:</b> Defects in HSD3B2 are the cause of adrenal hyperplasia type 2 (AH2) [MIM:201810]. AH2 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). In AH2, virilization is much less marked or does not occur. AH2 is frequently lethal in early life. Note=Mild HSD3B2 deficiency in hyperandrogenic females is associated with characteristic traits of polycystic ovary syndrome, such as insulin resistance and luteinizing hormon hypersecretion.
	<b>DISEASE:</b> Defects in HSD3B2 are the cause of adrenal hyperplasia type 2 (AH2) [MIM:201810]. AH2 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). In AH2, virilization is much less marked or does not occur. AH2 is frequently lethal in early life. Note=Mild HSD3B2 deficiency in hyperandrogenic females is associated with





for 30min; Antibody incubation with (HSD3B2) Polyclonal Antibody, Unconjugated
(SL16552R) at 1:400 overnight at 4°C, followed by operating according to SP
Kit(Rabbit) (sp-0023) instructions and DAB staining.

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