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HSD3B2 Rabbit pAb

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Catalog # AP94340

Product Information

Application	WB
Reactivity	Mouse
Host	Rabbit
Clonality	Polyclonal
Calculated MW	40 KDa
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from mouse HSD3B2
Epitope Specificity	151-250/372
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SUBCELLULAR LOCATION	Endoplasmic reticulum membrane. Mitochondrion membrane.
SIMILARITY	Belongs to the 3-beta-HSD family.
SUBUNIT	Expressed in adrenal gland, testis and ovary.
DISEASE	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
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	Predicted to enable 3-beta-hydroxy-delta5-steroid dehydrogenase activity and steroid delta-isomerase activity. Predicted to be involved in several processes, including hippocampus development; response to corticosterone; and steroid hormone biosynthetic process. Predicted to be located in several cellular components, including intercellular bridge; mitochondrial envelope; and nucleolus. Predicted to be active in cytoplasm and intracellular membrane-bounded organelle. Is expressed in liver. Human ortholog(s) of this gene implicated in hypertension and hypospadias. Orthologous to human HSD3B1 (hydroxy-delta-5-steroid dehydrogenase, 3 beta- and steroid delta-isomerase 1) and HSD3B2 (hydroxy-delta-5-steroid dehydrogenase, 3 beta- and steroid delta-isomerase 2). [provided by Alliance of Genome Resources, Apr 2022] 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. Tissue Specificity : 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. DISEASE : 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 Similarity : Belongs to the 3-beta-HSD family. SWISS: P26439 Gene ID : 3284 Human Gene ID : 3284 Database links : Entrez Gene: 3284 Human Entrez Gene: 15493 Mouse Entrez Gene: 29632 Rat SwissProt: P26439 Human SwissProt: P26149 Mouse

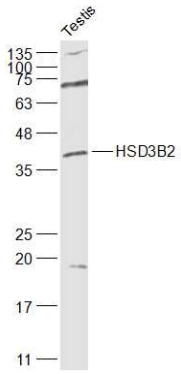
Additional Information

Target/Specificity	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.
Dilution	WB=1:500-2000
Format	0.01M TBS(pH7.4) with 1% BSA, 0.09% (W/V) sodium azide and 50% Glyce
Storage	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.

Background

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

Images



Sample: Testis (Mouse) Lysate at 40 ug Primary: Anti-HSD3B2 (AP94340) at 1/1000 dilution Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution Predicted band size: 40 kD Observed band size: 40 kD