– DATASHEET -

Host: Rabbit

Clonality: Polyclonal

151-250/372.

cycles.

GenelD: 15493 Target: HSD3B2

Concentration: 1mg/ml

[Primary Antibody]

HSD3B2 Rabbit pAb

www.bioss.com.cn sales@bioss.com.cn techsupport@bioss.com.cn 400-901-9800 Isotype: IgG Applications: WB (1:500-2000) Reactivity: Mouse Predicted 40 kDa **Immunogen:** KLH conjugated synthetic peptide derived from mouse HSD3B2: MW.: Purification: affinity purified by Protein A Subcellular Location: Cytoplasm Storage: 0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw Background: 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 deltaisomerase 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'

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

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Rat SwissProt: P26439 Human SwissProt: P26149 Mouse

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VALIDATION IMAGES



Sample: Testis (Mouse) Lysate at 40 ug Primary: Anti-HSD3B2 (bs-24205R) at 1/1000 dilution Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution Predicted band size: 40 kD Observed band size: 40 kD