SANTA CRUZ BIOTECHNOLOGY, INC.

AKR1D1 (H-70): sc-134968

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

AKR1D1 (aldo-keto reductase family 1 member D1), also known as δ 4-3-oxo-steroid 5 β-reductase (3o5bred) or steroid 5 β-reductase (SRD5B1), is responsible for catalyzing bile acid intermediates and steroid hormones possessing a δ 4-3-one structure to 5 β reduced metabolites. The AKR family of proteins are soluble NADPH oxidoreductases. They play important roles in the metabolism of drugs, carcinogens and reactive aldehydes. AKR1D1 is highly expressed in liver, colon and testis. Substrates for AKR1D1 include Testosterone, andro-stenedione, Progesterone, 17 α-hydroxyprogesterone and the bile acid intermediates 7 α-hydroxy-4-cholesten-3-one and 7 α, 12 α-dihydroxy-4-cholesten-3-one. A deficiency in AKR1D1 may be involved in hepatic dysfunction.

REFERENCES

1. Kondo, K.H., et al. 1994. Cloning and expression of cDNA of human δ 4-3-oxosteroid 5 β-reductase and substrate specificity of the expressed enzyme. Eur. J. Biochem. 219: 357-363.
2. Clayton, P.T., et al. 1996. δ 4-3-oxosteroid 5 β-reductase deficiency: failure of ursodeoxycholic acid treatment and response to chenodeoxycholic acid plus cholic acid. Gut 38: 623-628.
3. Sumazaki, R., et al. 1997. Gene analysis in δ 4-3-oxosteroid 5 β-reductase deficiency. Lancet 349: 329.
4. Charbonneau, A., et al. 1999. Assignment of steroid 5 β-reductase (SRD5B1) and its pseudogene (SRD5BP1) to human chromosome bands 7q32→q33 and 1q23→q25, respectively, by *in situ* hybridization. Cytogenet. Cell Genet. 84: 105-106.
5. Charbonneau, A., et al. 2001. Genomic organization of a human 5 β-reductase and its pseudogene and substrate selectivity of the expressed enzyme. Biochim. Biophys. Acta 1517: 228-235.
6. Lemonde, H.A., et al. 2003. Mutations in SRD5B1 (AKR1D1), the gene encoding δ 4-3-oxosteroid 5 β-reductase, in hepatitis and liver failure in infancy. Gut 52: 1494-1499.
7. Gonzales, E., et al. 2004. SRD5B1 (AKR1D1) gene analysis in δ 4-3-oxosteroid 5 β-reductase deficiency: evidence for primary genetic defect. J. Hepatol. 40: 716-718.
8. Sheehan, P.M., et al. 2005. 5 β-dihydroprogesterone and steroid 5 β-reductase decrease in association with human parturition at term. Mol. Hum. Reprod. 11: 495-501.

CHROMOSOMAL LOCATION

Genetic locus: AKR1D1 (human) mapping to 7q33; Akr1d1 (mouse) mapping to 6 B1.

SOURCE

AKR1D1 (H-70) is a rabbit polyclonal antibody raised against amino acids 198-267 mapping within an internal region of AKR1D1 of human origin.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

AKR1D1 (H-70) is recommended for detection of AKR1D1 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

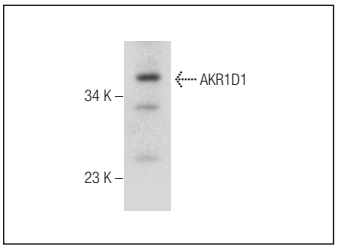
AKR1D1 (H-70) is also recommended for detection of AKR1D1 in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for AKR1D1 siRNA (h): sc-61964, AKR1D1 siRNA (m): sc-61965, AKR1D1 shRNA Plasmid (h): sc-61964-SH, AKR1D1 shRNA Plasmid (m): sc-61965-SH, AKR1D1 shRNA (h) Lentiviral Particles: sc-61964-V and AKR1D1 shRNA (m) Lentiviral Particles: sc-61965-V.

Molecular Weight of AKR1D1: 37 kDa.

Positive Controls: GA-10 whole cell lysate, mouse liver extract: sc-2256 or rat liver extract: sc-2395.

DATA



AKR1D1 (H-70): sc-134968. Western blot analysis of AKR1D1 expression in GA-10 whole cell lysate.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.