

CYP17A1 (N-18): sc-46085

BACKGROUND

The cytochrome P450 proteins are monooxygenases that catalyze many reactions involved in drug metabolism and synthesis of cholesterol, steroids and other lipids. P450 enzymes are classified into subfamilies, such as CYP1A, CYP2A, CYP2C, CYP2D, CYP4A14, CYP7A, CYP7B, CYP8B, CYP11A, CYP17A1, CYP19 and CYP27A, based on sequence similarities. CYP17A (17 α -hydroxylase/17,20-lyase) is important for the conversion of pregnenolone and progesterone to dehydroepiandrosterone (DHEA) and androstenedione. In this process, it catalyzes both the 17- α -hydroxylation and the 17,20-lyase reaction. CYP17A1 is crucial during sexual development, both during fetal development and during puberty, and is intracellularly regulated by cAMP levels. Defects in the CYP17A1 gene, which encodes for the protein, may cause adrenal hyperplasia type V (AH-V) which is characterized by hypokalemia and hypertension. Male patients affected by AH-V do not undergo normal sexual differentiation and develop female external genitalia and do not undergo pubertal development.

REFERENCES

1. Yanase, T., et al. 1992. Molecular basis of apparent isolated 17,20-lyase deficiency: compound heterozygous mutations in the C-terminal region (Arg(496)—Cys, Gln(461)—Stop) actually cause combined 17 α -hydroxylase/17,20-lyase. *Biochim. Biophys. Acta* 1139: 275-279.
2. Ahlgren, R., et al. 1992. Compound heterozygous mutations (Arg 239—stop, Pro 342—Thr) in the CYP17 (P45017 α) gene lead to ambiguous external genitalia in a male patient with partial combined 17 α -hydroxylase/17,20-lyase deficiency. *J. Clin. Endocrinol. Metab.* 74: 667-672.
3. Monno, S., et al. 1993. Mutation of histidine 373 to leucine in cytochrome P450c17 causes 17 α -hydroxylase deficiency. *J. Biol. Chem.* 268: 25811-25817.
4. Fardella, C.E., et al. 1994. Point mutation of Arg440 to His in cytochrome P450c17 causes severe 17 α -hydroxylase deficiency. *J. Clin. Endocrinol. Metab.* 79: 160-164.

CHROMOSOMAL LOCATION

Genetic locus: CYP17A1 (human) mapping to 10q24.32; Cyp17a1 (mouse) mapping to 19 C3.

SOURCE

CYP17A1 (N-18) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of CYP17A1 of human origin.

PRODUCT

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

Blocking peptide available for competition studies, sc-46085 P, (100 μ g peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

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

APPLICATIONS

CYP17A1 (N-18) is recommended for detection of CYP17A1 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), 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).

CYP17A1 (N-18) is also recommended for detection of CYP17A1 in additional species, including equine.

Suitable for use as control antibody for CYP17A1 siRNA (h): sc-45641, CYP17A1 siRNA (m): sc-45642, CYP17A1 shRNA Plasmid (h): sc-45641-SH, CYP17A1 shRNA Plasmid (m): sc-45642-SH, CYP17A1 shRNA (h) Lentiviral Particles: sc-45641-V and CYP17A1 shRNA (m) Lentiviral Particles: sc-45642-V.

Molecular Weight of CYP17A1: 55 kDa.

Positive Controls: SW-13 cell lysate: sc-24778, ES-2 cell lysate: sc-24674 or HeLa nuclear extract: sc-2120.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048. 2) Immunofluorescence: use donkey anti-goat IgG-FITC: sc-2024 (dilution range: 1:100-1:400) or donkey anti-goat IgG-TR: sc-2783 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

SELECT PRODUCT CITATIONS

1. Mostaghel, E., et al. 2010. Intraprostatic steroidogenic enzymes-letter. *Cancer Res.* 70: 8247-8249; author reply 8249-8250.
2. Hwang, D.Y., et al. 2011. CYP17A1 intron mutation causing cryptic splicing in 17 α -hydroxylase deficiency. *PLoS ONE* 6: e25492.
3. Schonemann, M.D., et al. 2012. Expression of P450c17 in the human fetal nervous system. *Endocrinology* 153: 2494-2505.

RESEARCH USE

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

PROTOCOLS

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


 MONOS
 Satisfaction
 Guaranteed

Try **CYP17A1 (D-12): sc-374244** or **CYP17A1 (G-4): sc-376711**, our highly recommended monoclonal alternatives to CYP17A1 (N-18).