SANTA CRUZ BIOTECHNOLOGY, INC.

CYP1A1 (H-70): sc-20772



BACKGROUND

P450 enzymes constitute a family of monooxygenase enzymes that are involved in the metabolism of a wide array of endogenous and xenobiotic compounds. Several P450 enzymes have been classified by sequence similarities as members of the CYP1A and CYP2A subfamilies. NADPH cytochrome P450 reductase is a microsomal enzyme responsible for the transfer of electrons from NADPH to cytochrome P450 enzymes during the P450 catalytic cycle. NADPH cytochrome P450 reductase is localized to the endoplasmic reticulum where it is also able to transfer electrons to heme oxygenase and cytochrome β 5. NADPH cytochrome P450 reductase is structurally related to two separate flavoprotein families, ferredoxin nucleotide reductase (FNR) and flavodoxin. Electron transfer of NADPH cytochrome P450 reductase requires the binding of two flavin cofactors, FAD and FMN, to the FNR and flavodoxin domains, respectively.

CHROMOSOMAL LOCATION

Genetic locus: CYP1A1 (human) mapping to 15q24.1; Cyp1a1 (mouse) mapping to 9 B.

SOURCE

CYP1A1 (H-70) is a rabbit polyclonal antibody raised against amino acids 246-315 mapping to an internal region of CYP1A1 of human origin.

PRODUCT

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

APPLICATIONS

CYP1A1 (H-70) is recommended for detection of CYP1A1 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for CYP1A1 siRNA (h): sc-41483, CYP1A1 siRNA (m): sc-41484, CYP1A1 shRNA Plasmid (h): sc-41483-SH, CYP1A1 shRNA Plasmid (m): sc-41484-SH, CYP1A1 shRNA (h) Lentiviral Particles: sc-41483-V and CYP1A1 shRNA (m) Lentiviral Particles: sc-41484-V.

Molecular Weight of CYP1A1: 56 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200 or CYP1A1 (h): 293T Lysate: sc-114027.

STORAGE

Store at 4° C, **D0 NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

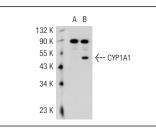
PROTOCOLS

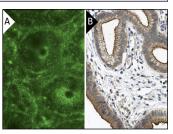
See our web site at www.scbt.com or our catalog for detailed protocols and support products.

RESEARCH USE

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

DATA





CYP1A1 (H-70): sc-20772. Western blot analysis of CYP1A1 expression in non-transfected: sc-117752 (**A**) and human CYP1A1 transfected: sc-114027 (**B**) 293T whole cell lysates.

CYP1A1 (H-70) sc-20772. Immunofluorescence staining of normal mouse intestine frozen section showing cytoplasmic staining (**A**). Immunoperoxidase staining of formalin fixed, paraffin-embedded human gall bladder tissue showing cytoplasmic and membrane staining of glandular cells magnification. Kindly provided by The Swedish Human Protein Atlas (HPA) program (**B**).

SELECT PRODUCT CITATIONS

- Zhang, S., et al. 2003. Flavonoids as aryl hydrocarbon receptor agonists/antagonists: effects of structure and cell context. Environ. Health Perspect. 111: 1877-1882.
- Wehbe, H., et al. 2006. Pifithrin-α enhances chemosensitivity by a p38 mitogen-activated protein kinase-dependent modulation of the eukaryotic initiation factor 4E in malignant cholangiocytes. J. Pharmacol. Exp. Ther. 319: 1153-1161.
- 3. Du, L., et al. 2006. Differentiation-specific factors modulate epidermal CYP1-4 gene expression in human skin in response to retinoic acid and classic aryl hydrocarbon receptor ligands. J. Pharmacol. Exp. Ther. 319: 1162-1171.
- Chang, H., et al. 2006. Preferential induction of CYP1A1 and CYP1B1 in CCSP-positive cells. Toxicol. Sci. 89: 205-213.
- Dougherty, E.J. and Pollenz, R.S. 2008. Analysis of Ah receptor-ARNT and Ah receptor-ARNT2 complexes *in vitro* and in cell culture. Toxicol Sci. 103: 191-206.
- Stoeger, T., et al. 2009. Deducing *in vivo* toxicity of combustion-derived nanoparticles from a cell-free oxidative potency assay and metabolic activation of organic compounds. Environ. Health Perspect. 117: 54-60.
- Dewa, Y., et al. 2009. Molecular expression analysis of β-naphthoflavoneinduced hepatocellular tumors in rats. Toxicol. Pathol. 37: 446-455.
- 8. Weems, J.M. and Yost, G.S. 2010. 3-Methylindole metabolites induce lung CYP1A1 and CYP2F1 enzymes by AhR and non-AhR mechanisms, respectively. Chem. Res. Toxicol. 23: 696-704.
- Terao, M., et al. 2011. Induction of miR-21 by retinoic acid in estrogen receptor-positive breast carcinoma cells: biological correlates and molecular targets. J. Biol. Chem. 286: 4027-4042.
- Oleaga, C., et al. 2011. CYP1A1 is overexpressed upon incubation of breast cancer cells with a polyphenolic cocoa extract. Eur. J. Nutr. E-published.