SANTA CRUZ BIOTECHNOLOGY, INC.

GAPDH (A-14): sc-20358

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Glyceraldehyde-3-phosphate dehydrogenase (GAPDH), also called uracil DNA glycosylase, catalyzes the reversible oxidative phosphorylation of glyceraldehyde-3-phosphate in the presence of inorganic phosphate and nicotinamide adenine dinucleotide (NAD), an important energy-yielding step in carbohydrate metabolism. While GAPDH has long been recognized as playing an integral role in glycolysis, additional functions of GAPDH include acting as an uracil DNA glycosylase, activating transcription, binding RNA and involvement in nuclear RNA export, DNA replication and DNA repair. Expression of GAPDH is upregulated in liver, lung and prostate cancers. GAPDH translocates to the nucleus during apoptosis. GAPDH complexes with neuronal proteins implicated in human neurodegenerative disorders including the β-Amyloid pre-cursor, Huntingtin and other triplet repeat neuronal disorder proteins.

CHROMOSOMAL LOCATION

Genetic locus: GAPDH (human) mapping to 12p13.31; Gapdh (mouse) mapping to 6 F3.

SOURCE

GAPDH (A-14) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the C-terminus of GAPDH 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.

Blocking peptide available for competition studies, sc-20358 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

GAPDH (A-14) is recommended for detection of GAPDH 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).

GAPDH (A-14) is also recommended for detection of GAPDH in additional species, including equine, canine, bovine, porcine, avian and feline.

Suitable for use as control antibody for GAPDH siRNA (h): sc-35448, GAPDH siRNA (m): sc-35449, GAPDH shRNA Plasmid (h): sc-35448-SH, GAPDH shRNA Plasmid (m): sc-35449-SH, GAPDH shRNA (h) Lentiviral Particles: sc-35448-V and GAPDH shRNA (m) Lentiviral Particles: sc-35449-V.

Molecular Weight of GAPDH: 37 kDa.

Positive Controls: GAPDH (h3): 293T Lysate: sc-113887, A549 cell lysate: sc-2413 or KNRK whole cell lysate: sc-2214.

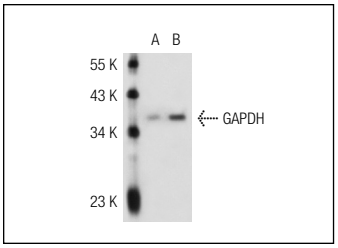
STORAGE

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

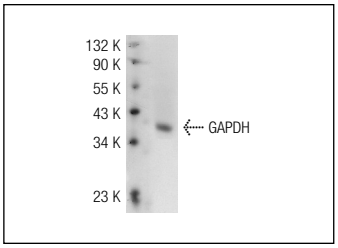
RESEARCH USE

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

DATA



GAPDH (A-14): sc-20358. Western blot analysis of GAPDH expression in non-transfected: sc-117752 (**A**) and human GAPDH transfected: sc-113887 (**B**) 293T whole cell lysates.



GAPDH (A-14): sc-20358. Western blot analysis of GAPDH expression in A459 whole cell lysate.

SELECT PRODUCT CITATIONS

1. Schroeder-Gloeckler, J.M., et al. 2007. CCAAT/enhancer-binding protein β deletion reduces adiposity, hepatic steatosis, and diabetes in Lepr(db/db) mice. J. Biol. Chem. 282: 15717-15729.
2. Lincová, E., et al. 2009. Multiple defects in negative regulation of the PKB/Akt pathway sensitise human cancer cells to the antiproliferative effect of non-steroidal anti-inflammatory drugs. Biochem. Pharmacol. 78: 561-572.
3. Lee, C.H., et al. 2010. Overexpression and activation of the α9-nicotinic receptor during tumorigenesis in human breast epithelial cells. J. Natl. Cancer Inst. 102: 1322-1335.
4. Huang, C.S., et al. 2010. Long-term ethanol exposure causes human liver cancer cells to become resistant to mitomycin C treatment through the inactivation of bad-mediated apoptosis. Mol. Carcinog. 49: 728-738.
5. Chen, C.S., et al. 2011. Nicotine-induced human breast cancer cell proliferation attenuated by garcinol through down-regulation of the nicotinic receptor and cyclin D3 proteins. Breast Cancer Res. Treat. 125: 73-87.
6. Staršíchová, A., et al. 2012. TGF-β1 signaling plays a dominant role in the crosstalk between TGF-β1 and the aryl hydrocarbon receptor ligand in prostate epithelial cells. Cell. Signal. 24: 1665-1676.
7. Rahman, S.M., et al. 2012. CCAAT/enhancer-binding protein β (C/EBPβ) expression regulates dietary-induced inflammation in macrophages and adipose tissue in mice. J. Biol. Chem. 287: 34349-34360.