SANTA CRUZ BIOTECHNOLOGY, INC.

ACO2 (A-22): sc-130677

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

ACO2, also referred to as aconitate hydratase, citrate hydrolyase or aconitase, is an iron-sulfur hydrolyase that catalyzes the non-limiting interconversion of citrate and isocitrate in the tricarboxylic acid cycle. It is expressed in the mitochondria and maintains a citrate:isocitrate ratio of approximately 10:1. ACO2 contains a redox-sensitive iron-sulfur cluster that exists in two states: active (Fe4S4) and inactive (Fe3S4). ACO2 activity is dependent on the state of this cluster as well as the presence of two conserved cysteine residues. In normal prostate epithelial cells ACO2 activity is prevented due to the high levels of zinc inhibiting the enzyme. In these citrate-producing epithelial cells citrate oxidation is impaired, allowing citrate to accumulate and exhibit a citrate:isocitrate ratio of approximately 30:1. In malignant prostate cells zinc is unable to accumulate, therefore ACO2 activity resumes and citrate is oxidized.

REFERENCES

1. Rafferty, S.P., et al. 1996. Inhibition of hemoglobin expression by heterologous production of nitric oxide synthase in the K-562 erythroleukemic cell line. Blood 88: 1070-1078.
2. Juang, H.H. 2004. Nitroprusside stimulates mitochondrial aconitase gene expression through the cyclic adenosine 3',5'-monosphosphate signal transduction pathway in human prostate carcinoma cells. Prostate 61: 92-102.
3. Liang, L.P. and Patel, M. 2004. Iron-sulfur enzyme mediated mitochondrial superoxide toxicity in experimental Parkinson's disease. J. Neurochem. 90: 1076-1084.
4. Yu, Z., et al. 2006. Characterization of the mitochondrial aconitase promoter and the identification of transcription factor binding. Prostate 66: 1061-1069.
5. Beasley, C.L., et al. 2006. Proteomic analysis of the anterior cingulate cortex in the major psychiatric disorders: evidence for disease-associated changes. Proteomics 6: 3414-3425.
6. Hunzinger, C., et al. 2006. Comparative profiling of the mammalian mitochondrial proteome: multiple aconitase-2 isoforms including N-formylkynurenine modifications as part of a protein biomarker signature for reactive oxidative species. J. Proteome Res. 5: 625-633.

CHROMOSOMAL LOCATION

Genetic locus: ACO2 (human) mapping to 22q13.2; Aco2 (mouse) mapping to 15 E1.

SOURCE

ACO2 (A-22) is a purified rabbit polyclonal antibody raised against a peptide mapping within an internal region of ACO2 of human origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

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

APPLICATIONS

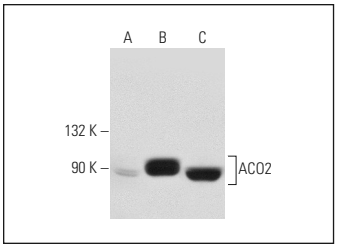
ACO2 (A-22) is recommended for detection of ACO2 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), 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 ACO2 siRNA (h): sc-61936, ACO2 siRNA (m): sc-61937, ACO2 shRNA Plasmid (h): sc-61936-SH, ACO2 shRNA Plasmid (m): sc-61937-SH, ACO2 shRNA (h) Lentiviral Particles: sc-61936-V and ACO2 shRNA (m) Lentiviral Particles: sc-61937-V.

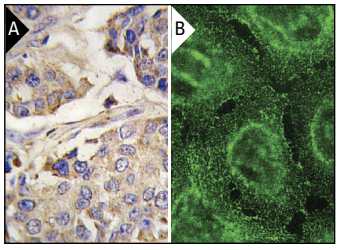
Molecular Weight of ACO2: 82 kDa.

Positive Controls: ACO2 (h): 293T Lysate: sc-127922, mouse heart extract: sc-2254 or K-562 whole cell lysate: sc-2203.

DATA



ACO2 (A-22): sc-130677. Western blot analysis of ACO2 expression in non-transfected 293T: sc-117752 (**A**), human ACO2 transfected 293T: sc-127922 (**B**) and K-562 (**C**) whole cell lysates.



ACO2 (A-22): sc-130677. Immunoperoxidase staining of formalin-fixed, paraffin-embedded human breast carcinoma tissue showing cytoplasmic localization (**A**). Immunofluorescence staining of methanol-fixed HeLa cells showing membrane and cytoplasmic localization **(B)**.

SELECT PRODUCT CITATIONS

1. Nordin, A., et al. 2011. Tissue-specific splicing of ISCU results in a skeletal muscle phenotype in myopathy with lactic acidosis, while complete loss of ISCU results in early embryonic death in mice. Hum. Genet. 129: 371-378.
2. Dai, D.F., et al. 2012. Mitochondrial proteome remodelling in pressure overload-induced heart failure: the role of mitochondrial oxidative stress. Cardiovasc. Res. 93: 79-88.
3. Lettieri Barbato, D., et al. 2013. Proline oxidase-adipose triglyceride lipase pathway restrains adipose cell death and tissue inflammation. Cell Death Differ. 21: 113-123.

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.