SANTA CRUZ BIOTECHNOLOGY, INC.

ACYP2 (G-5): sc-398251

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The formation of stable highly organized protein aggegrates, known as amyloid fibrils, is associated with several debilitating human diseases, including Alzheimer's disease, Parkinson's disease, and Creutzfeldt-Jakob disease. In each of these conditions, a peptide or protein that is normally soluble accumulates into insoluble fibrils. Muscle acylphosphatase (AcP) has emerged as a significant model system to study protein misfolding and aggregation. It is particularly suitable for these studies because muscle AcP is a small, simple protein of only 98 amino acids consisting of a five-stranded antiparallel β-sheet and two parallel α-helices. Mutations in the muscle AcP between residues 16-31 and 87-98, which includes its phosphate binding site at Arg-23, significantly increases the rate of aggregation. These mutations correlate with changes in the hydrophobicity of AcP and a conversion of the α-helical structures to β-sheets. Therefore, a reduction in the net charge of a protein may be a key determinant in some forms of protein deposition diseases.

REFERENCES

1. Serpell, L.C., et al. 1997. The molecular basis of amyloidosis. Cell. Mol. Life Sci. 53: 871-887.
2. Chiti, F., et al. 2000. Mutational analysis of the propensity for amyloid formation by a globular protein. EMBO J. 19: 1441-1449.
3. Chiti, F., et al. 2001. Reduction of the amyloidogenicity of a protein by specific binding of ligands to the native conformation. Protein Sci. 10: 879-886.
4. Taddei, N., et al. 2001. Folding and aggregation are selectively influenced by the conformational preferences of the α-helices of muscle acylphosphatase. J. Biol. Chem. 276: 37149-37154.
5. Chiti, F., et al. 2002. Studies of the aggregation of mutant proteins *in vitro* provide insights into the genetics of amyloid diseases. Proc. Natl. Acad. Sci. USA 99: 16419-16426.
6. Chiti, F., et al. 2002. Kinetic partitioning of protein folding and aggregation. Nat. Struct. Biol. 9: 137-143.

CHROMOSOMAL LOCATION

Genetic locus: ACYP2 (human) mapping to 2p16.2.

SOURCE

ACYP2 (G-5) is a mouse monoclonal antibody raised against amino acids 1-99 representing full length ACYP2 of human origin.

PRODUCT

Each vial contains 200 µg IgG_1 kappa light chain in 1.0 ml of 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

ACYP2 (G-5) is recommended for detection of muscle ACYP2 of human origin by Western Blotting (starting dilution 1:100, 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).

Suitable for use as control antibody for ACYP2 siRNA (h): sc-38900, ACYP2 shRNA Plasmid (h): sc-38900-SH and ACYP2 shRNA (h) Lentiviral Particles: sc-38900-V.

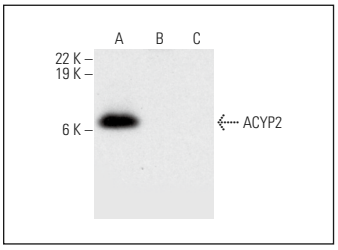
Molecular Weight of ACYP2: 11 kDa.

Positive Controls: human skeletal muscle extract: sc-363776.

RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use m-IgGκ BP-FITC: sc-516140 or m-IgGκ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz® Mounting Medium: sc-24941 or UltraCruz® Hard-set Mounting Medium: sc-359850.

DATA



ACYP2 (G-5): sc-398251. Western blot analysis of ACYP2 expression in human skeletal muscle (**A**), mouse skeletal muscle (**B**) and rat skeletal muscle (**C**) tissue extracts. Note lack of reactivity with mouse and rat ACYP2 in lanes **B** and **C**.

SELECT PRODUCT CITATIONS

1. Degl'Innocenti, D., et al. 2019. Oxadiazon affects the expression and activity of aldehyde dehydrogenase and acylphosphatase in human striatal precursor cells: a possible role in neurotoxicity. Toxicology 411: 110-121.

RESEARCH USE

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

PROTOCOLS

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