SANTA CRUZ BIOTECHNOLOGY, INC.

Na^+/K^+-ATPase α3 (F-1): sc-374050

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The ubiquitously expressed sodium/potassium-ATPase (Na^+/K^+-ATPase) exists as a oligomeric plasma membrane complex that couples the hydrolysis of one molecule of ATP to the importation of three Na^+ ions and two K^+ ions against their respective electrochemical gradients. As a member of the P-type family of ion motives, Na^+/K^+-ATPase plays a critical role in maintaining cellular volume, resting membrane potential and Na^+-coupled solute transport. Multiple isoforms of three subunits, α, β and γ, comprise the Na^+/K^+-ATPase oligomer. The α subunit contains the binding sites for ATP and the cations; the glycosylated β subunit ensures correct folding and membrane insertion of the α subunits. The small γ subunit co-localizes with the α subunit in nephron segments, where it increases the affinity of Na^+/K^+-ATPase for ATP. The β subunit, but not the γ subunit, is essential for normal activity of Na^+/K^+-ATPase.

CHROMOSOMAL LOCATION

Genetic locus: ATP1A3 (human) mapping to 19q13.2; Atp1a3 (mouse) mapping to 7 A3.

SOURCE

Na^+/K^+-ATPase α3 (F-1) is a mouse monoclonal antibody specific for an epitope mapping between amino acids 419-446 within an internal region of Na^+/K^+-ATPase α3 of human origin.

PRODUCT

Each vial contains 200 µg IgG_3 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-374050 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% stabilizer protein).

APPLICATIONS

Na^+/K^+-ATPase α3 (F-1) is recommended for detection of Na^+/K^+-ATPase α3 of mouse, rat and 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).

Na^+/K^+-ATPase α3 (F-1) is also recommended for detection of Na^+/K^+-ATPase α3 in additional species, including equine, canine and porcine.

Suitable for use as control antibody for Na^+/K^+-ATPase α3 siRNA (h): sc-36012, Na^+/K^+-ATPase α3 siRNA (m): sc-36013, Na^+/K^+-ATPase α3 shRNA Plasmid (h): sc-36012-SH, Na^+/K^+-ATPase α3 shRNA Plasmid (m): sc-36013-SH, Na^+/K^+-ATPase α3 shRNA (h) Lentiviral Particles: sc-36012-V and Na^+/K^+-ATPase α3 shRNA (m) Lentiviral Particles: sc-36013-V.

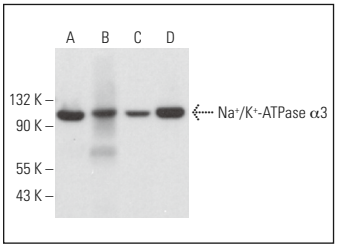
Molecular Weight of Na^+/K^+-ATPase α3: 113 kDa.

Positive Controls: mouse brain extract: sc-2253, rat brain extract: sc-2392 or human brain extract: sc-364375.

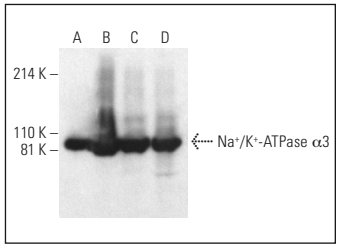
STORAGE

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

DATA



Na^+/K^+-ATPase α3 (F-1): sc-374050. Western blot analysis of Na^+/K^+-ATPase α3 expression in rat brain (**A**), mouse brain (**B**), human brain (**C**) and human cerebellum (**D**) tissue extracts.



Na^+/K^+-ATPase α3 (F-1): sc-374050. Western blot analysis of Na^+/K^+-ATPase α3 expression in human brain (**A**), mouse brain (**B**), rat cerebellum (**C**) and rat brain (**D**) tissue extracts. Detection reagent used: m-IgG_3 BP-HRP: sc-533670.

SELECT PRODUCT CITATIONS

1. Heinzen, E.L., et al. 2012. *De novo* mutations in ATP1A3 cause alternating hemiplegia of childhood. Nat. Genet. 44: 1030-1034.
2. Müller, L.G., et al. 2015. Effects of diene valepotriates from *Valeriana glechomifolia* on Na^+/K^+-ATPase activity in the cortex and hippocampus of mice. Planta Med. 81: 200-207.
3. Paciorkowski, A.R., et al. 2015. Novel mutations in ATP1A3 associated with catastrophic early life epilepsy, episodic prolonged apnea, and postnatal microcephaly. Epilepsia 56: 422-430.
4. Liu, M., et al. 2016. A novel bufalin derivative exhibited stronger apoptosis-inducing effect than bufalin in A549 lung cancer cells and lower acute toxicity in mice. PLoS ONE 11: e0159789.
5. Lazarov, E., et al. 2020. Comparative analysis of alternating hemiplegia of childhood and rapid-onset dystonia-parkinsonism ATP1A3 mutations reveals functional deficits, which do not correlate with disease severity. Neurobiol. Dis. 143: 105012.
6. Arystarkhova, E., et al. 2021. Misfolding, altered membrane distributions, and the unfolded protein response contribute to pathogenicity differences in Na,K-ATPase ATP1A3 mutations. J. Biol. Chem. 296: 100019.

RESEARCH USE

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