SANTA CRUZ BIOTECHNOLOGY, INC.

NMNAT-2 (B-10): sc-515206

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

NMNAT proteins are essential cofactors involved in the fundamental processes of cell metabolism. They belong to the eukaryotic NMN adenylyltransferase family. NMNATs participate in the synthesis of NAD^+ by catalyzing the condensation of nicotinamide mononucleotide and ATP. The presence of magnesium and other divalent cations increases their enzymatic activity. The interaction of NMNATs with nuclear proteins is likely to be modulated by phosphorylation. NMNAT proteins contain at least three potential phosphorylation sites and may act as substrates for nuclear kinases. NMNAT-2 (nicotinamide mononucleotide adenylyltransferase 2) is a 307 amino acid protein that is highly expressed in the brain, especially in the cerebrum, cerebellum, occipital lobe, frontal lobe, temporal lobe and putamen. It is also detected at lower levels in the heart and skeletal muscle. Two isoforms exists due to alternate splicing.

CHROMOSOMAL LOCATION

Genetic locus: NMNAT2 (human) mapping to 1q25.3; Nmnat2 (mouse) mapping to 1 G3.

SOURCE

NMNAT-2 (B-10) is a mouse monoclonal antibody raised against amino acids 208-307 mapping at the C-terminus of NMNAT-2 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.

NMNAT-2 (B-10) is available conjugated to agarose (sc-515206 AC), 500 µg/ 0.25 ml agarose in 1 ml, for IP; to HRP (sc-515206 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-515206 PE), fluorescein (sc-515206 FITC), Alexa Fluor® 488 (sc-515206 AF488), Alexa Fluor® 546 (sc-515206 AF546), Alexa Fluor® 594 (sc-515206 AF594) or Alexa Fluor® 647 (sc-515206 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-515206 AF680) or Alexa Fluor® 790 (sc-515206 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

Alexa Fluor® is a trademark of Molecular Probes, Inc., Oregon, USA

APPLICATIONS

NMNAT-2 (B-10) is recommended for detection of NMNAT-2 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).

Suitable for use as control antibody for NMNAT-2 siRNA (h): sc-62693, NMNAT-2 siRNA (m): sc-62694, NMNAT-2 shRNA Plasmid (h): sc-62693-SH, NMNAT-2 shRNA Plasmid (m): sc-62694-SH, NMNAT-2 shRNA (h) Lentiviral Particles: sc-62693-V and NMNAT-2 shRNA (m) Lentiviral Particles: sc-62694-V.

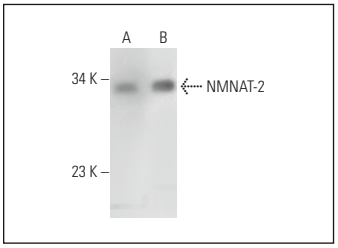
Molecular Weight of NMNAT-2: 34 kDa.

Positive Controls: human brain extract: sc-364375, NIH/3T3 whole cell lysate: sc-2210 or Neuro-2A whole cell lysate: sc-364185.

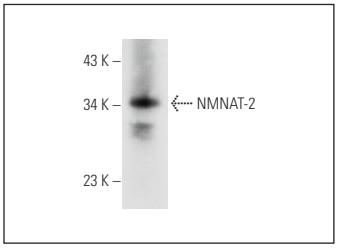
STORAGE

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

DATA



NMNAT-2 (B-10): sc-515206. Western blot analysis of NMNAT-2 expression in NIH/3T3 (**A**) and Neuro-2A (**B**) whole cell lysates.



NMNAT-2 (B-10): sc-515206. Western blot analysis of NMNAT-2 expression in human brain tissue extract.

SELECT PRODUCT CITATIONS

1. Qi, J., et al. 2018. Downregulated SIRT6 and upregulated NMNAT2 are associated with the presence, depth and stage of colorectal cancer. Oncol. Lett. 16: 5829-5837.
2. Geisler, S., et al. 2019. Vincristine and bortezomib use distinct upstream mechanisms to activate a common SARM1-dependent axon degeneration program. JCI Insight 4: e129920.
3. Fan, M., et al. 2020. Overexpression of the histidine triad nucleotide-binding protein 2 protects cardiac function in the adult mice after acute myocardial infarction. Acta Physiol. 228: e13439.
4. Ko, K.W., et al. 2020. SARM1 acts downstream of neuroinflammatory and necroptotic signaling to induce axon degeneration. J. Cell Biol. 219: e201912047.
5. Niu, J., et al. 2020. Coupled control of distal axon integrity and somal responses to axonal damage by the palmitoyl acyltransferase ZDHHC17. Cell Rep. 33: 108365.
6. Hughes, R.O., et al. 2021. Small molecule SARM1 inhibitors recapitulate the $SARM1^{-/-}$ phenotype and allow recovery of a metastable pool of axons fated to degenerate. Cell Rep. 34: 108588.
7. Sasaki, Y., et al. 2021. Nicotinic acid mononucleotide is an allosteric SARM1 inhibitor promoting axonal protection. Exp. Neurol. 345: 113842.
8. Zhang, C., et al. 2021. Atg7 knockout alleviated the axonal injury of neuro-2a cells induced by Tri-Ortho-Cresyl phosphate. Neurotox. Res. 39: 1076-1086.
9. Parsons, R.B., et al. 2022. α-synucleinopathy reduces NMNAT3 protein levels and neurite formation that can be rescued by targeting the NAD^+ pathway. Hum. Mol. Genet. 31: 2918-2933.

RESEARCH USE

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