

α -synuclein (3H2897): sc-69977

BACKGROUND

The synuclein family members, including α -synuclein (also designated NACP for non- β -Amyloid component) and β -synuclein, are predominantly expressed in the brain and are speculated to be involved in synaptic regulation and neuronal plasticity. α -synuclein is localized to neuronal cell bodies and synapses. α -synuclein was first identified as a component of Alzheimer's disease amyloid plaques. Abnormal platelet function in Alzheimer's disease has been demonstrated. During megakaryocytic differentiation, α -synuclein has been found to be upregulated, while β -synuclein is downregulated, indicating that coordinate expression of synucleins may be important during hematopoietic cell differentiation. A mutant form of α -synuclein has been found in patients with early onset Parkinson's disease.

CHROMOSOMAL LOCATION

Genetic locus: SNCA (human) mapping to 4q22.1; Snca (mouse) mapping to 6 B3.

SOURCE

α -synuclein (3H2897) is a mouse monoclonal antibody raised against recombinant α -synuclein of human origin.

PRODUCT

Each vial contains 50 μ g IgG₁ in 500 μ l of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

α -synuclein (3H2897) is recommended for detection of α -synuclein of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500).

Suitable for use as control antibody for α -synuclein siRNA (h): sc-29619, α -synuclein siRNA (m): sc-42286, α -synuclein shRNA Plasmid (h): sc-29619-SH, α -synuclein shRNA Plasmid (m): sc-42286-SH, α -synuclein shRNA (h) Lentiviral Particles: sc-29619-V and α -synuclein shRNA (m) Lentiviral Particles: sc-42286-V.

Molecular Weight of α -synuclein: 19 kDa.

Positive Controls: SH-SY5Y cell lysate: sc-3812, α -synuclein (h): 293T lysate: sc-111729 or IMR-32 cell lysate: sc-2409.

STORAGE

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

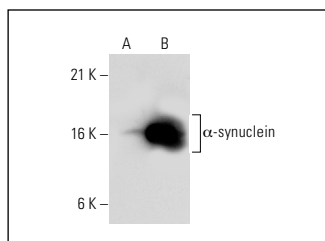
PROTOCOLS

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

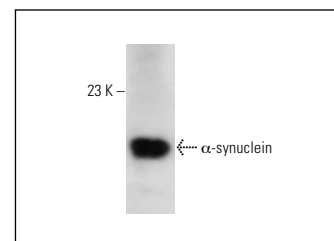
RESEARCH USE

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

DATA



α -synuclein (3H2897): sc-69977. Western blot analysis of α -synuclein expression in non-transfected: sc-117752 (A) and human α -synuclein transfected: sc-111729 (B) 293T whole cell lysates.



α -synuclein (3H2897): sc-69977. Western blot analysis of α -synuclein expression in IMR-32 whole cell lysate.

SELECT PRODUCT CITATIONS

- Schiess, M.C., et al. 2010. CSF from Parkinson disease patients differentially affects cultured microglia and astrocytes. *BMC Neurosci.* 11: 151.
- Deleersnijder, A., et al. 2011. Comparative analysis of different peptidyl-prolyl isomerases reveals FK506-binding protein 12 as the most potent enhancer of α -synuclein aggregation. *J. Biol. Chem.* 286: 26687-26701.
- Hoseini, S.G., et al. 2012. Regulatory T-cell profile in early and late lesions of cutaneous leishmaniasis due to *Leishmania major*. *J. Res. Med. Sci.* 17: 513-518.
- Liu, Y., et al. 2015. Environment-contact administration of rotenone: a new rodent model of Parkinson's disease. *Behav. Brain Res.* 294: 149-161.
- Xuan, Q., et al. 2016. Post-translational modifications of α -synuclein contribute to neurodegeneration in the colon of elderly individuals. *Mol. Med. Rep.* 13: 5077-5083.
- Kim, J.S., et al. 2017. α -synuclein in the colon and premotor markers of Parkinson disease in neurologically normal subjects. *Neurol. Sci.* 38: 171-179.
- Erustes, A.G., et al. 2018. Overexpression of α -synuclein in an astrocyte cell line promotes autophagy inhibition and apoptosis. *J. Neurosci. Res.* 96: 160-171.
- Wang, Z.H., et al. 2018. BDNF inhibits neurodegenerative disease-associated asparaginyl endopeptidase activity via phosphorylation by Akt. *JCI Insight* 3: e99007.
- Rahimi-Balaei, M., et al. 2019. Loss of prostatic acid phosphatase and α -synuclein cause motor circuit degeneration without altering cerebellar patterning. *PLoS ONE* 14: e0222234.



See **α -synuclein (211): sc-12767** for α -synuclein antibody conjugates, including AC, HRP, FITC, PE, and Alexa Fluor® 488, 546, 594, 647, 680 and 790.