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Alpha-Synuclein Rabbit pAb

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Catalog # AP94696

Product Information

Application	WB, E
Host	Rabbit
Clonality	Polyclonal
Calculated MW	14 KDa
Physical State	Liquid
Immunogen	Recombinant full length Alpha-Synuclein
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SUBCELLULAR LOCATION	Cytoplasm. Membrane. Nucleus. Cell junction, synapse. Note=Membrane-bound in dopaminergic neurons.
SIMILARITY	Belongs to the synuclein family.
SUBUNIT	Soluble monomer which can form filamentous aggregates. Interacts with UCHL1 (By similarity). Interacts with phospholipase D and histones. Subcellular Location : Cytoplasm. Membrane. Nucleus. Cell junction, synapse. Note=Membrane-bound in dopaminergic neurons.
Post-translational modifications	Phosphorylated, predominantly on serine residues. Phosphorylation by CK1 appears to occur on residues distinct from the residue phosphorylated by other kinases. Phosphorylation of Ser-129 is selective and extensive in synucleinopathy lesions. In vitro, phosphorylation at Ser-129 promoted insoluble fibril formation. Phosphorylated on Tyr-125 by a PTK2B-dependent pathway upon osmotic stress.Hallmark lesions of neurodegenerative synucleinopathies contain alpha-synuclein that is modified by nitration of tyrosine residues and possibly by dityrosine cross-linking to generated stable oligomers.Ubiquitinated. The predominant conjugate is the diubiquitinated form (By similarity).Acetylation at Met-1 seems to be important for proper folding and native oligomeric structure.
DISEASE	Note=Genetic alterations of SNCA resulting in aberrant polymerization into fibrils, are associated with several neurodegenerative diseases (synucleinopathies). SNCA fibrillar aggregates represent the major non A-beta component of Alzheimer disease amyloid plaque, and a major component of Lewy body inclusions. They are also found within Lewy body (LB)-like intraneuronal inclusions, glial inclusions and axonal spheroids in neurodegeneration with brain iron accumulation type 1.Parkinson disease 1 (PARK1) [MIM:168601]: A complex neurodegenerative disorder characterized by bradykinesia, resting tremor, muscular rigidity and postural instability. Additional features are characteristic postural abnormalities, dysautonomia, dystonic cramps, and dementia. The pathology of Parkinson disease involves the loss of dopaminergic neurons in the substantia nigra and the presence of Lewy bodies (intraneuronal accumulations of aggregated proteins), in surviving neurons in various areas of the brain. The disease is progressive and usually manifests after the age of 50 years, although early-onset cases (before 50 years) are known. The majority of the cases are sporadic suggesting a

multifactorial etiology based on environmental and genetic factors. However, some patients present with a positive family history for the disease. Familial forms of the disease usually begin at earlier ages and are associated with atypical clinical features. Note=The disease is caused by mutations affecting the gene represented in this entry. [DISEASE] Parkinson disease 4 (PARK4) [MIM:605543]: A complex neurodegenerative disorder with manifestations ranging from typical Parkinson disease to dementia with Lewy bodies. Clinical features include parkinsonian symptoms (resting tremor, rigidity, postural instability and bradykinesia), dementia, diffuse Lewy body pathology, autonomic dysfunction, hallucinations and paranoia. Note=The disease is caused by mutations affecting the gene represented in this entry.Dementia Lewy body (DLB) [MIM:127750]: A neurodegenerative disorder characterized by mental impairment leading to dementia, parkinsonism, fluctuating cognitive function, visual hallucinations, falls, syncopal episodes, and sensitivity to neuroleptic medication. Brainstem or cortical intraneuronal accumulations of aggregated proteins (Lewy bodies) are the only essential pathologic features. Patients may also have hippocampal and neocortical senile plaques, sometimes in sufficient number to fulfill the diagnostic criteria for Alzheimer disease. Note=The disease is caused by mutations affecting the gene represented in this entry.

Important Note This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

Background Descriptions Alpha-synuclein is a member of the synuclein family, which also includes beta- and gamma-synuclein. Synucleins are abundantly expressed in the brain and alpha- and beta-synuclein inhibit phospholipase D2 selectively. SNCA may serve to integrate presynaptic signaling and membrane trafficking. Defects in SNCA have been implicated in the pathogenesis of Parkinson disease. SNCA peptides are a major component of amyloid plaques in the brains of patients with Alzheimer's disease. Alternatively spliced transcripts encoding different isoforms have been identified for this gene. [provided by RefSeq, Feb 2016].

Additional Information

Target/Specificity Expressed principally in brain but is also expressed in low concentrations in all tissues examined except in liver. Concentrated in presynaptic nerve terminals.

Dilution WB=1:200-1000,ELISA=1:5000-10000

Format 0.01M TBS(pH7.4) with 1% BSA, 0.09% (W/V) sodium azide and 50% Glyce

Storage Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Background

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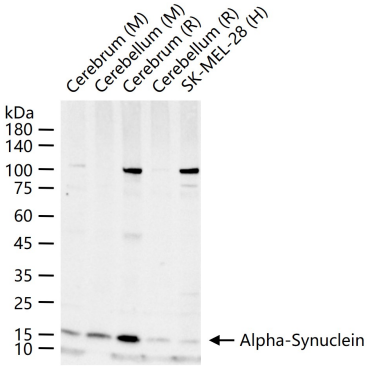
Images

25 ug total protein per lane of various lysates (see on figure) probed with Alpha-Synuclein polyclonal antibody, unconjugated (AP94696) at 1:1000 dilution and 4°C



overnight incubation. Followed by conjugated secondary antibody incubation at r.t. for 60 min.