

Product datasheet

Recombinant Human Alpha-synuclein (mutated E46K) protein ab51188

[2 References](#) [1 Image](#)

Description

Product name	Recombinant Human Alpha-synuclein (mutated E46K) protein
Purity	> 95 % SDS-PAGE. Alpha Synuclein E46K was overexpressed in <i>E. coli</i> and purified to apparent homogeneity by using conventional column chromatography techniques.
Expression system	Escherichia coli
Accession	<u>P37840</u>
Protein length	Full length protein
Animal free	No
Nature	Recombinant
Species	Human
Sequence	MDVFMKGLSK/ AKEGVVAAAE/ KTKQGVAEAA/ GKTKEGVLVY/ GSKTK[K]GVVH/GVATVAEKTQ/EQVTNVGGAV/VT GVTAVAQK/TVEGAGSIAA/ ATGFVKKDQL/GKNEEGAPQE/GILEDM PVDP/DNEAYEMPSE/EGYQDYEP EA
Predicted molecular weight	14 kDa
Amino acids	1 to 140
Modifications	mutated E46K

Specifications

Our **Abpromise guarantee** covers the use of **ab51188** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications SDS-PAGE

Form Liquid

Additional notes

Recent studies have shown that this mutant(E46K) of alpha-synuclein causes Parkinson and Lewy Body dementia(DLB).

Preparation and Storage

Stability and Storage

Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
pH: 7.50
Constituents: 0.316% Tris HCl, 0.58% Sodium chloride

General Info

Function

May be involved in the regulation of dopamine release and transport. Induces fibrillization of microtubule-associated protein tau. Reduces neuronal responsiveness to various apoptotic stimuli, leading to a decreased caspase-3 activation.

Tissue specificity

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

Involvement in disease

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
Parkinson disease 4
Dementia Lewy body

Sequence similarities

Belongs to the synuclein family.

Domain

The 'non A-beta component of Alzheimer disease amyloid plaque' domain (NAC domain) is involved in fibrils formation. The middle hydrophobic region forms the core of the filaments. The C-terminus may regulate aggregation and determine the diameter of the filaments.

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.

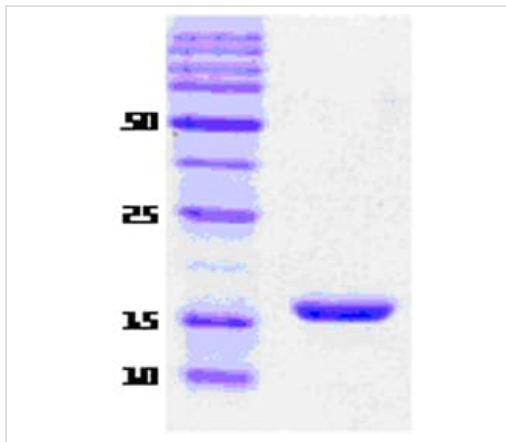
Ubiquitinated. The predominant conjugate is the diubiquitinated form.

Acetylation at Met-1 seems to be important for proper folding and native oligomeric structure.

Cellular localization

Cytoplasm, cytosol. Membrane. Nucleus. Cell junction, synapse. Secreted. Membrane-bound in dopaminergic neurons.

Images



ab51188 in 15%SDS-PAGE

SDS-PAGE - Recombinant Human Alpha-synuclein
(mutated E46K) protein (ab51188)

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