3050 Spruce Street, St. Louis, MO 63103 USA
Tel: (800) 521-8956 (314) 771-5765 Fax: (800) 325-5052 (314) 771-5757
email: techservice@sial.com sigma-aldrich.com

Product Information

Anti-α-Synuclein
produced in rabbit, IgG fraction of antiserum

Catalog Number **S3062**

Product Description

Anti-α-Synuclein is produced in rabbit using as immunogen a synthetic peptide corresponding to a sequence near the C-terminus of human α-synuclein, amino acids 111-132 with C-terminally added lysine, conjugated to KLH. This sequence is highly conserved (90% homology) in mouse and rat α-synuclein. This sequence has no homology with β- and γ-synuclein. Whole antiserum is purified to provide the IgG fraction of antiserum.

Anti-α-Synuclein recognizes human and rat α-synuclein. Applications include the detection and localization of α-synuclein by immunoblotting (19 kDa) and immunohistochemistry. Staining of α-synuclein in immunoblotting is specifically inhibited by the immunizing peptide.

The synucleins are a family of soluble presynaptic proteins that are abundant in neurons and include α-synuclein, β-synuclein, and γ-synuclein.[1-5] The functions of the synuclein superfamily of proteins are poorly understood, however several lines of evidence suggest potential roles in synaptic function and neural plasticity.[4, 5] Human α-synuclein (also known as the non-amyloid component of plaques precursor protein or NACP) is a 140-amino acid polypeptide that is encoded by a gene on chromosome 4.[6] It was originally isolated from plaques of Alzheimer's disease (AD) brains as a 19 kDa protein precursor of the highly hydrophobic 35-amino acid peptide, nonamyloid component (NAC) of plaques.[3] The NAC can self-aggregate into fibrils and induce aggregation of the β-amyloid peptide.

α-Synuclein is highly abundant in presynaptic terminals,[7] and is a major component of Lewy bodies (LBs). LBs are neuronal cytoplasmic inclusions that are found in diverse neurodegenerative disorders, including a subtype of Alzheimer's disease with abundant neocortical LBs, known as Lewy body variant of Alzheimer's disease, as well as in diffuse Lewy body disease, and in Parkinson's disease.[8] Pathogenic point mutations in the α-synuclein gene are linked to familial Parkinson's disease (PD) in rare kindreds.[9] However, most neurodegenerative disorders with LBs are associated with abnormal accumulation of wild-type, α-synuclein. Further, LBs have been described in familial AD caused by presenilin and amyloid precursor protein gene mutations and in Down syndrome.[10] Deletion of the α-synuclein gene in mice results in functional deficits of the nigrostriatal dopamine system.[11] Neuronal over-expression of wild-type human α-synuclein in mice resulted in progressive accumulation of α-synuclein in neurons, associated with loss of dopaminergic terminals in the basal ganglia and with motor impairment, suggesting that α-synuclein may play a role in PD and related conditions.[12]

α-Synuclein is expressed primarily in brain, but is also expressed in low levels in all tissues examined except liver.

Reagent

Supplied in a solution of 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

Store at –20 °C. For continuous use, the product may be stored at 2-8 °C for up to one month. For prolonged storage, freeze in working aliquots at –20 °C. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

Product Profile

Immunoblotting: a minimum working antibody dilution of 1:1,000 is determined using an extract of rat brain homogenate.

Immunohistochemistry: a minimum working antibody dilution of 1:200 is determined using formalin-fixed/paraffin-embedded, formic acid treated sections of Alzheimer's disease brain.

Note: In order to obtain the best results in various techniques and preparations, we recommend determining optimal working dilutions by titration.

References

1. Maroteaux, L., et al., *J. Neurosci*., **8**, 2804 (1988).
2. Maroteaux, L., and Scheller, R.H., *Mol. Brain. Res*., **11**, 335 (1991).
3. Ueda, K., et al., *Proc. Natl. Acad. Sci. USA*, **90**, 11282 (1993).
4. Jakes, R., et al., *FEBS Lett*., **345**, 27 (1994).
5. Clayton, D.F., and George, JM., *Trends Neurosci*., **21**, 249 (1998).
6. Chen, X., et al., *Genomics*, **26**, 425 (1995).
7. Iwai, A., et al., *Neuron*, **14**, 467 (1995).
8. Spillantini, M.G., et al., *Proc. Natl. Acad. Sci. USA*, **95**, 6469 (1998).
9. Polymeropoulos, M.H., et al., *Science*, **276**, 2045 (1997).
10. Lippa, C.F., et al., *Am. J. Pathol*., **153**, 1365 (1998).
11. Abeliovich, A., et al., *Neuron*, **25**, 239 (2001).
12. Masliah, E., et al., *Science*, **287**, 1265 (2000).

MG,KAA,PHC 07/09-1