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ProductInformation

α-Synuclein A53T

human, recombinant, expressed in *E.coli*

Catalog Number **S 1071**
Storage Temperature: –20 °C

Product Description

α-Synuclein, also known as the non-amyloid component of plaques precursor protein or NACP, is a 140-amino-acid protein (apparent molecular weight 19-20 kDa) encoded by a simple gene consisting of six exons on human chromosome 4.[1] The physiological role of α-synuclein is not clear. In the search for its function it was found that α-synuclein induces polymerization of tubulin into microtubules.[2] In addition, α-synuclein was found to function in the modulation of dopamine transporter function, regulating the synaptic tone of dopamine.[3] Disruption of this function can ultimately lead to neurodegeneration of nerve terminals.

α-Synuclein is highly abundant in presynaptic terminals[4] and is a major component of Lewy bodies (LBs). LBs are neuronal cytoplasmic inclusions that are found in diverse neurodegenerative disorders. The deposition of α-synuclein as fibrillary aggregates in neurons or glial cells is a hallmark lesion in a subset of neurodegenerative disorders. These disorders include Parkinson's disease (PD), dementia with Lewy bodies (filamentous inclusions), Lewy body variant of Alzheimer's disease, and multiple system atrophy.[2] Pathogenic point mutations in the α-synuclein gene, like the Ala^{30}-Pro (A30P) and Ala^{53}- Thr (A53T) missense mutations, are linked to familial Parkinson's disease.[5] However, most neurodegenerative disorders involving LBs are associated with abnormal accumulation of wild-type α-synuclein. Mice expressing A53T human α-synuclein, but not wild-type or the A30P variants, develop adult-onset neurodegenerative disease with a progressive motoric dysfunction leading to death.[6] Deletion of the α-synuclein gene in mice results in functional deficits of the nigrostriatal dopamine system.[7] 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 Parkinson Disease and related conditions.[8]

Purity: ≥90% (SDS-PAGE)

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

The product is shipped on dry ice and stored at –20°C. Reconstitute with water to ~1 mg/ml. Store the reconstituted solution in working aliquots at –20 °C. The reconstituted product is stable for at least 1 year.

References:

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3. Sidhu, A., et al., α-Synuclein regulation of the dopaminergic transporter: a possible role in the pathogenesis of Parkinson's disease. *FEBS Lett.*, **565**, 1-5 (2004).
4. Iwai, A., et al., The precursor protein of non-A β component of Alzheimer's disease amyloid is a presynaptic protein of the central nervous system. *Neuron*, **14**, 467-475 (1995).
5. Polymeropoulos, M.H., et al., Mutation in the α-synuclein gene identified in families with Parkinson's disease. *Science*, **276**, 2045-2047 (1997).
6. Lee, M.K., et al., Human α-synuclein-harboring familial Parkinson's disease-linked Ala-53- Thr mutation causes neurodegenerative disease with α-synuclein aggregation in transgenic mice. *Proc. Natl. Acad. Sci. USA,* **99**, 8968-8973 (2002).

7. Abeliovich, A., et al., Mice lacking α-synuclein display functional deficits in the nigrostriatal dopamine system. *Neuron*, **25**, 239-251(2000).

8. Masliah, E., et al., Dopaminergic loss and inclusion body formation in α-synuclein mice: implications for neurodegenerative disorders. *Science*, **287**, 1265-1269 (2000).

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