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Alpha Synuclein TNG (A53T, S87N, N103G) Mutant Monomers



Discovery through Partnership | Excellence through Quality

Human Recombinant Alpha Synuclein TNG
(A53T, S87N, N103G) Mutant Monomers
Catalog No. SPR-503

Product Name

Alpha Synuclein TNG (A53T, S87N, N103G) Mutant Monomers

Description

Human Recombinant Alpha Synuclein TNG (A53T, S87N, N103G) Mutant Monomers

Applications

WB, SDS PAGE, In vitro Assay

Concentration

Lot/batch specific. See included datasheet.

Conjugates

No tag

Nature

Recombinant

Species

Human

Expression System

E. coli

Amino Acid Sequence

MDVFMKGLSKAKEGVVAAAEEKTKQGVAEAAGKTKEGVLYVGSKTKEGVVHGVTTVAEKTKEQVTNVGGAWVTGVTAVAQ
KTVEGAGNIAAATGFVKKDQLGKGEEGAPQEGILEDMPVPDNEAYEMPSEEGYQDYEPEA

Purity

>95%

Other Resources

Protein Length

Full length (1 - 140 aa)

Field Of Use

Not for use in humans. Not for use in diagnostics or therapeutics. For in vitro research use only.

Properties

Storage Buffer

1X PBS pH7.4

Storage Temperature

-80°C

Shipping Temperature

Dry Ice. Shipping note: Product will be shipped separately from other products purchased in the same order.

Purification

Ion-exchange Purified

Cite This Product

Human Recombinant Alpha Synuclein TNG (A53T, S87N, N103G) Mutant Monomers (StressMarq Biosciences Inc., Victoria BC CANADA, Catalog # SPR-503)

Certificate Of Analysis

Protein certified >95% pure on SDS-PAGE & Nanodrop analysis, low endotoxin

Biological Description

Alternative Names

Alpha synuclein protein, Alpha-synuclein protein, Non-A beta component of AD amyloid protein, Non-A4 component of amyloid precursor protein, NACP protein, SNCA protein, NACP protein, PARK1 protein, SYN protein, Parkinson's disease familial 1 Protein, Alpha Synuclein TNG

Research Areas

Alzheimer's Disease, Neurodegeneration, Neuroscience, Parkinson's Disease, Synuclein, Tangles & Tau, Multiple System Atrophy

Swiss Prot

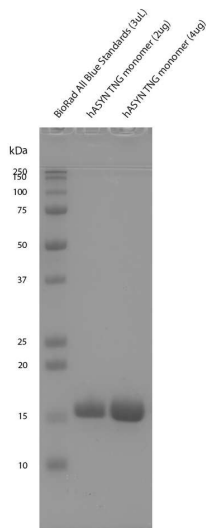
Scientific Background

Human alpha synuclein TNG mutant (HuTNG) is a triple mutant containing Ala53 mutated to the equivalent mouse residue Thr53, Ser87 mutated to the equivalent mouse residue Asn87, and Asn103 mutated to the equivalent mouse residue Gly103, effectively making it a human-mouse chimeric protein. Despite sequence differences at only seven residues, or 5% of the total 140 amino acids, the aggregation rate of wild-type mouse α -syn (MsWT) is faster than wild-type human α -syn (HuWT) in vitro. In wild-type mouse models, MsWT fibrils are more efficient than HuWT fibrils at inducing endogenous mouse α -syn pathology (1). A53T or S87N substitutions in human α -syn substantially accelerate fibrilization rates in vitro (2,3). Chimeric HuTNG fibrils show enhanced induction of α -syn pathology greater than both HuWT and MsWT fibrils after single unilateral injection into the dorsal striatum in mice (4). Therefore, HuTNG is a good construct for inducing robust endogenous α -syn seeding and pathology in wild-type mice.

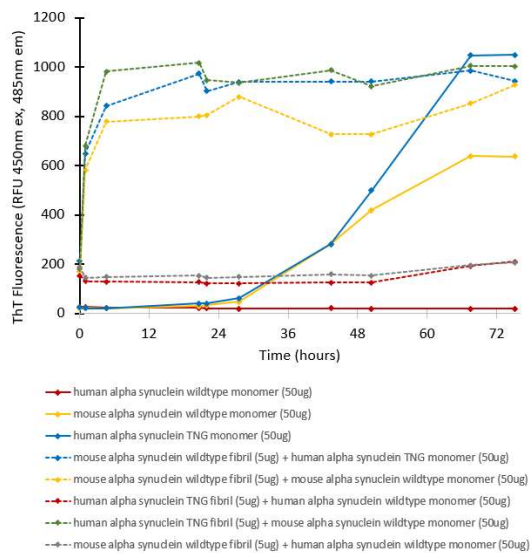
References

1. Masuda-Suzukake et al. 2013. Prion-like Spreading of Pathological α -synuclein in Brain. *Brain*. <https://doi.org/10.1093/brain/awt037>
2. Kang, K. et al. 2011. The A53T Mutation is Key in Defining the Differences in the Aggregation Kinetics of Human and Mouse α -synuclein. *JACS*. <https://doi.org/10.1021/ja203979j>
3. Ohgita, T. et al. 2023. Intramolecular Interaction Kinetically Regulates Fibril Formation by Human and Mouse Alpha-Synuclein. *Sci Rep* <https://doi.org/10.1038/s41598-023-38070-4>
4. Luk, K., C. et al. 2016. Molecular and Biological Compatibility with Host Alpha-Synuclein Influences Fibril Pathogenicity. *Cell Rep*. <https://doi.org/10.1016/j.celrep.2016.08.053>

Product Images



SDS-PAGE of purified human alpha synuclein TNG (A53T, S87N, N103G) monomer (SPR-503) on a 12% Bis-Tris Gel. 2ug and 4ug of total protein were loaded into the respective lanes. Electrophoresis was run at 200V for 45 minutes with fixed voltage using 1X MES running buffer.



Fibril formation and seeding activity of human alpha synuclein TNG mutant measured by ThT in vitro. Human TNG mutant monomers (SPR-503) self-aggregate faster than human wild-type monomers. Human TNG mutant pre-formed fibrils (SPR-504) rapidly seed mouse wild-type monomers, but not human wild-type monomers, similar to the seeding pattern observed for mouse wild-type pre-formed fibrils.

Product Citations (0)

Currently there are no citations for this product.

Reviews

There are no reviews yet.