Data Sheet (Cat.No.TMPY-03884)

TargetMol

Beta-amyloid 39/Beta-APP39 Protein, Human, Recombinant (aa 672-710, His & GST)

General Information

Synonyms:	CTFgamma;PN-II;AAA;Aβ;PN2;APPI;AD1;amyloid beta (A4) precursor protein;CTFγ;ABETA;β-amyloid 39/β-APP39;ABPP;amyloid β (A4) precursor protein;CVAP
Protein Construction:	A DNA sequence encoding the amino acids (Asp672-Val710) of human Amyloid beta A4 protein (APP770) (P05067-1), corresponding to the Beta-amyloid protein 39, was fused with the N-terminal polyhistidine-tagged GST tag at the N-terminus. Predicted N terminal: Met
Species:	Human
Expression Host:	E. coli
Accession:	P05067-1
Molecular Weight:	32.2 kDa (predicted); 34 kDa (reducing conditions)

QC Testing

Biological Activity:	Activity testing is in progress. It is theoretically active, but we cannot guarantee it. If you require protein activity, we recommend choosing the eukaryotic expression version first.
Purity:	> 85 % as determined by SDS-PAGE
Endotoxin:	Please contact us for more information.
Formulation:	Supplied as sterile 50 mM Tris, 100 mM NaCl, 20% glycerol, 0.05% Tween 20, pH 9. 5.

Preparation and Storage

Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

Stability & Storage:

It is recommended to store the product under sterile conditions at -20°C to -80°C. Samples are stable for up to 12 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

Proteins are shipped with blue ice.

Protein Background

Amyloid precursor protein (APP) is a type I transmembrane protein expressed in many tissues and concentrated in the synapses of neurons, and is suggested as a regulator of synapse formation and neural plasticity. APP can be processed by two different proteolytic pathways. In one pathway, APP is cleaved by β- and γ-secretase to produce the amyloid-β-protein (Aβ, Abeta, beta-amyloid) which is the principal component of the amyloid plaques, the major pathological hallmark of Alzheimer's disease (AD), while in the other pathway, α-secretase is involved in the cleavage of APP whose product exerts antiamyloidogenic effect and prevention of the Aβ peptide formation. The aberrant accumulation of aggregated beta-amyloid peptides (Abeta) as plaques is a hallmark of AD

neuropathology and reduction of Abeta has become a leading direction of emerging experimental therapies for the disease. Besides this pathological function of Abeta, recently published data reveal that Abeta also has an essential physiological role in lipid homeostasis. Cholesterol increases Abeta production, and conversely A beta production causes a decrease in cholesterol synthesis. Abeta may be part of a mechanism controlling synaptic activity, acting as a positive regulator presynaptically and a negative regulator postsynaptically. The pathological accumulation of oligomeric Abeta assemblies depresses excitatory transmission at the synaptic level, but also triggers aberrant patterns of neuronal circuit activity and epileptiform discharges at the network level. Abeta-induced dysfunction of inhibitory interneurons likely increases synchrony among excitatory principal cells and contributes to the destabilization of neuronal networks. There is evidence that beta-amyloid can impair blood vessel function. Vascular beta-amyloid deposition, also known as cerebral amyloid angiopathy, is associated with vascular dysfunction in animal and human studies. Alzheimer disease is associated with morphological changes in capillary networks, and soluble beta-amyloid produces abnormal vascular responses to physiological and pharmacological stimuli.

Reference

Grimm MO, et al. (2007) Amyloid beta as a regulator of lipid homeostasis. Trends Mol Med. 13(8): 337-44.
Smith EE, et al. (2009) Beta-amyloid, blood vessels, and brain function. Stroke. 40(7): 2601-6.
Gouras GK, et al. (2010) Intraneuronal beta-amyloid accumulation and synapse pathology in Alzheimer's disease. Acta Neuropathol. 119(5): 523-41.
Palop JJ, et al. (2010) Amyloid-beta-induced neuronal dysfunction in Alzheimer's disease: from synapses toward neural networks. Nat Neurosci. 13(7): 812-8.

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