Data Sheet (Cat.No.TMPY-02221)

TargetMol

Beta-amyloid 42/Beta-APP42 Protein, Human, Recombinant (His & GST)

General Information

Synonyms:	CVAP;PN2;amyloid β (A4) precursor protein;Aβ;CTFγ;β-amyloid 42/β-APP42;AAA;ABPP;CTFgamma;PN-II;AD1;APPI;amyloid beta (A4) precursor protein;ABETA
Protein Construction:	A DNA sequence encoding the amino acids (Asp672-Ala713) of human Amyloid beta A4 protein (APP770) (P05067-1), corresponding to the Beta-amyloid protein 42, 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:	32.4 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:	> 80 % as determined by SDS-PAGE
Endotoxin:	Please contact us for more information.
Formulation:	Lyophilized from a solution filtered through a 0.22 μm filter, containing PBS, 10% glycerol, pH 7.4. Typically, a mixture containing 5% to 8% trehalose, mannitol, and 0.01% Tween 80 is incorporated as a protective agent before lyophilization.

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 recombinant proteins at -20°C to -80°C for future use. Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry 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.