

## Beta-amyloid/Beta-APP Protein, Human, Recombinant (hFc)

## General Information

Synonyms:	CTF $\gamma$ ; amyloid beta (A4) precursor protein; AAA; ABPP; CVAP; APPI; AD1; PN2; amyloid $\beta$ (A4) precursor protein; PN-II; ABETA; CTF $\gamma$ ; A $\beta$
Protein Construction:	A DNA sequence encoding the human APP-751 isoform (NP_958816.1) (Met 1-Leu 669) was expressed with the C-terminal fused Fc region of human IgG1. Predicted N terminal: Leu 18
Species:	Human
Expression Host:	HEK293 Cells
Accession:	P05067-8
Molecular Weight:	101 kDa (predicted); 150-160 kDa (reducing condition, due to glycosylation)

## QC Testing

Biological Activity:	Measured by its ability to inhibit trypsin cleavage of a fluorogenic peptide substrate, Mca-RPKPVE-Nval-WRK(Dnp)-NH <sub>2</sub> . The IC <sub>50</sub> value is < 1.2 nM.
Purity:	> 95 % as determined by SDS-PAGE
Endotoxin:	< 1.0 EU/ $\mu$ g of the protein as determined by the LAL method.
Formulation:	Lyophilized from a solution filtered through a 0.22 $\mu$ m filter, containing PBS, 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 &amp; 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  $\beta$ - and  $\gamma$ -secretase to produce the amyloid- $\beta$ -protein (A $\beta$ , 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,  $\alpha$ -secretase is involved in the cleavage of APP whose product exerts anti-amyloidogenic effect and prevention of the A $\beta$  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 Abeta 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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Tel: 781-999-4286 E\_mail: info@targetmol.com Address: 34 Washington Street, Wellesley Hills, MA 02481