

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



Catalog Number:PKSH031465

**Note:** Centrifuge before opening to ensure complete recovery of vial contents.

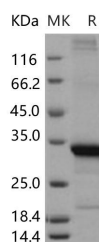
## Description

<b>Synonyms</b>	AAA;ABETA;ABPP;AD1;APPI;CTFgamma;CVAP;PN-II;PN2
<b>Species</b>	Human
<b>Expression Host</b>	E.coli
<b>Sequence</b>	Asp672-Ala713
<b>Accession</b>	P05067-1
<b>Calculated Molecular Weight</b>	32.4 kDa
<b>Observed molecular weight</b>	34 kDa
<b>Tag</b>	N-His-GST

## Properties

<b>Purity</b>	> 80 % as determined by reducing SDS-PAGE.
<b>Endotoxin</b>	Please contact us for more information.
<b>Storage</b>	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
<b>Shipping</b>	This product is provided as lyophilized powder which is shipped with ice packs.
<b>Formulation</b>	Lyophilized from sterile PBS, 10% glycerol, pH 7.4 Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
<b>Reconstitution</b>	Please refer to the printed manual for detailed information.

## Data



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## 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. Abeta may be part of a mechanism

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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. 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.

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