Elabscience®

Recombinant Human APP Protein(Trx Tag)

Catalog Number: PDEH100549

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

Description			
Species	Human		
Source	E.coli-derived Human APP protein Asp672-Asn770, with an N-terminal Trx		
Calculated MW	30.8 kDa		
Observed MW	35 kDa		
Accession	P05067-1		
Bio-activity	Not validated for activity		
Properties			
Purity	>90% as determined by reducing SDS-PAGE.		
Endotoxin	< 10 EU/mg of the protein as determined by the LAL method		
Storage	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 $< -20^{\circ}$ C for 3 months.		
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.		
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with 5% Trehalose and 5%		
	Mannitol.		
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of		
	0.5 mg/mL. Concentration is measured by UV-Vis.		

Data

kDa	м	R
80	-	
60	-	
40	-	
		-
30		
20		
20		

SDS-PAGE analysis of Human APP proteins, 2 µg/lane of Recombinant Human APP proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 30.8 KD

Background

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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 (A β , 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 $A\beta$ 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 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 oligometric 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 betaamyloid 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.