Amyloid Precursor/Beta-APP40 Protein, Human (His-GST)

Cat. No.:	HY-P75511
Synonyms:	Amyloid-beta precursor protein; APP; Protease Nexin II; PreA4
Species:	Human
Source:	E. coli
Accession:	P05067 (D672-V711)
Gene ID:	351
Molecular Weight:	Approximately 33 kDa

PROPERTIES

Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of 50 mM Tris, 500 mM NaCl, pH 7.5. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	APP (Amyloid Precursor Protein), also known as Protease Nexin-II, operates as a multifunctional cell surface receptor, exerting physiological effects on neurons that are crucial for neurite growth, neuronal adhesion, and axonogenesis. Its involvement in synaptogenesis is highlighted by the promotion of synaptic connections through interactions between APP molecules on adjacent cells. Beyond cell adhesion, APP plays a role in cell mobility and transcriptional regulation through protein-protein interactions. It can stimulate transcription activation by binding to APBB1-KAT5 and inhibit Notch signaling through interaction with Numb. Additionally, APP couples to apoptosis-inducing pathways, such as those mediated by G(o) and JIP, and inhibits G(o) alpha ATPase activity. Acting as a kinesin I membrane receptor, APP facilitates axonal transport of beta-secretase and presenilin 1, contributing to axonal anterograde cargo transport towards synapses. In the context of copper homeostasis, APP is involved in copper ion reduction and can induce neuronal death through copper-metallated interactions. Furthermore, APP regulates neurite outgrowth by binding to extracellular matrix components and possesses protease inhibitor activity through its BPTI domain-containing isoforms. The protein participates in the AGER-dependent pathway, activating p38 MAPK and inducing internalization of amyloid-beta peptide, leading to mitochondrial dysfunction. Additionally, APP provides Cu(2+) ions for GPC1, required for nitric oxide release and heparan sulfate degradation. It exhibits metal-chelating properties, reduces transient metals, and binds to lipoproteins, apolipoproteins, and HDL particles, thereby modulating metal-catalyzed oxidation. APP's intricate involvement in various cellular processes underscores its significance

	in both normal neuronal function and pathological conditions associated with neurodegenerative disorders.

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA