

beta-Amyloid 1-42 Antibody FITC Conjugated

Catalog No: #C00059F

Package Size: #C00059F 100ul

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Description

Product Name	beta-Amyloid 1-42 Antibody FITC Conjugated
Host Species	Rabbit
Clonality	Polyclonal
Isotype	IgG
Purification	Purified by Protein A.
Applications	Flow-Cyt IF
Species Reactivity	Hu Ms Rt
Immunogen Description	KLH conjugated synthetic peptide derived from human beta-Amyloid(35-42)
Conjugates	FITC
Target Name	beta-Amyloid 1-42
Other Names	AAA; AD1; PN2; ABPP; APPI; CVAP; ABETA; PN-II; CTFgamma; Amyloid beta A4 protein; APP; Alzheimer disease amyloid protein; Amyloid precursor protein; Beta-amyloid precursor protein; Cerebral vascular amyloid peptide; PreA4; Protease nexin-II; A4
Accession No.	Swiss-Prot#P05067NCBI Gene ID351
Cell Localization	Extracellular, Helical
Concentration	1mg ml
Formulation	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage	Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.

Application Details

Flow-Cyt=1:50-200 IF=1:50-200

Background

Functions as a cell surface receptor and performs physiological functions on the surface of neurons relevant to neurite growth, neuronal adhesion and axonogenesis. Involved in cell mobility and transcription regulation through protein-protein interactions. Can promote transcription activation through binding to APBB1-KAT5 and inhibits Notch signaling through interaction with Numb. Couples to apoptosis-inducing pathways such as those mediated by G(O) and JIP. Inhibits G(o) alpha ATPase activity (By similarity). Acts as a kinesin I membrane receptor, mediating the axonal transport of beta-secretase and presenilin 1. Involved in copper homeostasis oxidative stress through copper ion reduction. In vitro, copper-metallated APP induces neuronal death directly or is potentiated through Cu(2+)-mediated low-density lipoprotein oxidation. Can regulate neurite outgrowth through binding to components of the extracellular matrix such as heparin and collagen I and IV. The splice isoforms that contain the BPTI domain possess protease inhibitor activity. Induces a AGER-dependent pathway that involves activation of p38 MAPK, resulting in internalization of amyloid-beta peptide and leading to mitochondrial dysfunction in cultured cortical neurons. Provides Cu(2+) ions for GPC1 which are required for release of nitric oxide (NO) and subsequent degradation of the heparan sulfate chains on GPC1. Beta-amyloid peptides are lipophilic metal chelators with metal-reducing activity. Bind transient metals such as copper, zinc and iron. In vitro, can reduce Cu(2+) and Fe(3+) to Cu(+) and Fe(2+), respectively. Beta-amyloid 42 is a more effective reductant than beta-amyloid 40. Beta-amyloid peptides bind to lipoproteins and apolipoproteins E and J in the CSF and to HDL particles in plasma, inhibiting metal-catalyzed oxidation of lipoproteins. Beta-APP42 may activate mononuclear phagocytes in the brain and elicit inflammatory responses.

Note: This product is for in vitro research use only and is not intended for use in humans or animals.