SunLong Biotech Co.,LTD
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[Marker Primary Antibody]

Rabbit Anti-beta Amyloid 1-42/FITC Conjugated antibody

Catalog Number: SL0107R-FITC

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	Anti-beta Amyloid 1-42/FITC
Immunogen:	KLH conjugated synthetic peptide of human beta-Amyloid
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Applications:	Flow-Cyt=1:50-200 ICC=1:50-200 IF=1:50-200 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Human(predicted:Mouse,Rat,Chicken,Dog,Pig,Cow,Rabbit)
Subcellular Location:	The cell membrane
Background:	background: The cerebral and vascular plaques associated with Alzheimer's disease are mainly composed of Amyloid beta peptides. beta Amyloid is derived from cleavage of the Amyloid precursor protein and varies in length from 39 to 43 amino acids. beta Amyloid [1-40], beta Amyloid [1-42], and beta Amyloid [1-43] peptides result from cleavage of Amyloid precursor protein after residues 40, 42, and 43, respectively. The cleavage takes place by gamma-secretase during the last Amyloid precursor protein processing step. beta Amyloid [1-40], beta Amyloid [1-42], and beta Amyloid [1-43] peptides are major constituents of the plaques and tangles that occur in Alzheimer's disease. beta Amyloid antibodies and peptides have been developed as tools for elucidating the biology of Alzheimer's disease. **Function:** 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

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	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. 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. 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. Promotes both tau aggregation and TPK II-mediated phosphorylation. Interaction with overexpressed HADH2 leads to oxidative stress and neurotoxicity. Appicans elicit adhesion of neural cells to the extracellular matrix and may regulate neurite outgrowth in the brai. The gamma-CTF peptides as well as the caspase-cleaved peptides, including C31, are potent enhancers of neuronal apoptosis. N-APP binds TNFRSF21 triggering caspase activation and degeneration of both neuronal cell bodies (via caspase-3) and axons (via caspase-6). **Subunit:** Binds, via its C-terminus, to the PID domain of several cytoplasmic proteins, including APBB family members, the APBA family, MAPK8IP1, SHC1 and, NUMB and DAB. Binding to DAB1 inhibits its serine phosphorylation (By similarity). Also interacts with GPCR-like protein BPP, FPRL1, APPBP1, IB1, KNS2 (via its TPR domains), APPBP2 (via BaSS) and DDB1. In vitro, it binds MAPT via the MT-binding domains. Associates with microtubules in the presence of ATP and in a kinesin-dependent manner. Interacts, through a C-terminal domain, with GNAO1. Amyloid beta-42 binds CHRNA7 in hippocampal neurons. Beta-amyloid associates with HADH2.

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	Soluble APP binds, via its N-terminal head, to FBLN1. Interacts with CPEB1 and AGER. Interacts with ANKS1B and TNFRSF21. Interacts with ITM2B. Interacts with ITM2C. Interacts with IDE. Can form homodimers; this is promoted by heparin binding. **Subcellular Location:** Membrane; Single-pass type I membrane protein. Membrane, clathrin-coated pit. Note=Cell surface protein that rapidly becomes internalized via clathrin-coated pits. During maturation, the immature APP (N-glycosylated in the endoplasmic reticulum) moves to the Golgi complex where complete maturation occurs (O-glycosylated and sulfated). After alpha-secretase cleavage, soluble APP is released into the extracellular space and the C-terminal is internalized to endosomes and lysosomes. Some APP accumulates in secretory transport vesicles leaving the late Golgi compartment and returns to the cell surface. Gamma-CTF(59) peptide is located to both the cytoplasm and nuclei of neurons. It can be translocated to the nucleus through association with APBB1 (Fe65). Beta-APP42 associates with FRPL1 at the cell surface and the complex is then rapidly internalized. APP sorts to the basolateral surface in epithelial cells. During neuronal differentiation, the Thr-743 phosphorylated form is located mainly in growth cones, moderately in neurites and sparingly in the cell body. Casein kinase phosphorylation can occur either at the cell surface or within a post-Golgi compartment. **Tissue Specificity:** Expressed in all fetal tissues examined with highest levels in brain, kidney, heart and spleen. Weak expression in liver. In adult brain, highest expression found in the frontal lobe of the cortex and in the anterior perisylvian cortex-opercular gyri. Moderate expression in the cerebellar cortex, the posterior perisylvian cortex-opercular gyri and the temporal associated cortex. Weak expression found in the striate, extra-striate and motor cortices. Expressed in cerebrospinal fluid, and plasma. Isoform APP695 is the predominant form in neuronal tissue, isoform APP751 and isoform APP770 are widely expressed in non-neuronal cells. Isoform APP751 is the most abundant form in T-lymphocytes. Appican is expressed in astrocytes. **Post-translational modifications:** Proteolytically processed under normal cellular conditions. Cleavage either by alpha-secretase, beta-secretase or theta-secretase leads to generation and extracellular release of soluble APP peptides, S-APP-alpha and S-APP-beta, and the retention of corresponding membrane-anchored C-terminal fragments, C80, C83 and C99. Subsequent processing of C80 and C83 by gamma-secretase yields P3 peptides. This is the major secretory pathway and is non-amyloidogenic. Alternatively, presenilin/nicastrin-mediated gamma-secretase processing of C99 releases the

	amyloid beta proteins, amyloid-beta 40 (Abeta40) and amyloid-beta 42 (Abeta42), major components of amyloid plaques, and the cytotoxic C-terminal fragments, gamma-CTF(50), gamma-CTF(57) and gamma-CTF(59). Proteolytically cleaved by caspases during neuronal apoptosis. Cleavage at Asp-739 by either caspase-6, -8 or -9 results in the production of the neurotoxic C31 peptide and the increased production of beta-amyloid peptides. N- and O-glycosylated. O-linkage of chondroitin sulfate to the L-APP isoforms produces the APP proteoglycan core proteins, the appicans. The chondroitin sulfate chain of appicans contains 4-O-sulfated galactose in the linkage region and chondroitin sulfate E in the repeated disaccharide region. Phosphorylation in the C-terminal on tyrosine, threonine and serine residues is neuron-specific. Phosphorylation can affect APP processing, neuronal differentiation and interaction with other proteins. Phosphorylated on Thr-743 in neuronal cells by Cdc5 kinase and Mapk10, in dividing cells by Cdc2 kinase in a cell-cycle dependent manner with maximal levels at the G2/M phase and, in vitro, by GSK-3-beta. The Thr-743 phosphorylated form causes a conformational change which reduces binding of Fe65 family members. Phosphorylation on Tyr-757 is required for SHC binding. Phosphorylated in the extracellular domain by casein kinases on both soluble and membrane-bound APP. This phosphorylation is inhibited by heparin. Extracellular binding and reduction of copper, results in a corresponding oxidation of Cys-144 and Cys-158, and the formation of a disulfide bond. In vitro, the APP-Cu(+) complex in the presence of hydrogen peroxide results in an increased production of beta-amyloid-containing peptides. Trophic-factor deprivation triggers the cleavage of surface APP by beta-secretase to release sAPP-beta which is further cleaved to release an N-terminal fragment of APP (N-APP). Beta-amyloid peptides are degraded by IDE. DISEASE: Defects in APP are the cause of Alzheimer disease type 1 (AD1) [MIM:104300]. AD1 is a familial early-onset form of Alzheimer disease. It can be associated with cerebral amyloid angiopathy. Alzheimer disease is a neurodegenerative disorder characterized by progressive dementia, loss of cognitive abilities, and deposition of fibrillar amyloid proteins as intraneuronal neurofibrillary tangles, extracellular amyloid plaques and vascular amyloid deposits. The major constituent of these plaques is the neurotoxic amyloid-beta-APP 40-42 peptide (s), derived proteolytically from the transmembrane precursor protein APP by sequential secretase processing. The cytotoxic C-terminal fragments (CTFs) and the caspase-cleaved products such as C31 derived from APP, are also implicated in neuronal death.

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	Defects in APP are the cause of cerebral amyloid angiopathy APP-related (CAA-APP) [MIM:605714]. A hereditary localized amyloidosis due to amyloid-beta A4 peptide(s) deposition in the cerebral vessels. The principal clinical characteristics are recurrent cerebral and cerebellar hemorrhages, recurrent strokes, cerebral ischemia, cerebral infarction, and progressive mental deterioration. Patients develop cerebral hemorrhage because of the severe cerebral amyloid angiopathy. Parenchymal amyloid deposits are rare and largely in the form of pre-amyloid lesions or diffuse plaque-like structures. They are Congo red negative and lack the dense amyloid cores commonly present in Alzheimer disease. Some affected individuals manifest progressive aphasic dementia, leukoencephalopathy, and occipital calcifications. **Similarity:** Belongs to the APP family. Contains 1 BPTI/Kunitz inhibitor domain. **Database links:** **Entrez Gene:** 351 Human **Entrez Gene:** 11820 Mouse **Entrez Gene:** 54226 Rat Omim: 104760 Human **SwissProt:** P05067 Human **SwissProt:** P12023 Mouse **SwissProt:** P08592 Rat **Unigene:** 434980 Human **Unigene:** 277585 Mouse **Unigene:** 2104 Rat Important **Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications. β-Amyloid also called β-A4 protein, is a product of β-amyloid precursor protein (APP) cleaved by β- and γ-secretase, composed of 39-43 amino acid residues. It is the main component of amyloid protein. This protein forms insoluble deposits in the brain (senile plaques), thereby possessing the morphological features of AD, Down's syndrome and senile dementia. β-amyloid protein is derived from β-amyloid precursor protein，Is deposited as filamentous protein-like deposits extracellularly in brain tissue ， Is the main protein component of amyloid nodular neuritis lesions，Also deposited in nerve fibers。 In senile dementia Alzheimer's disease, it characteristically appears in the cerebral cortexβ-Senile plaques formed by amyloid protein deposition。 Mainly used for the detection of amyloid substances in senile plaques in brain tissue of patients with Alzheimer's disease。If necessary, treat paraffin tissue

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	sections with 98-100% formic acid for 2-3 minutes。

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