SunLong Biotech Co.,LTD
Tel: 0086-571-56623320 Fax:0086-571-56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com

Mouse Anti-beta-Amyloid (1-42)antibody

SL0107M

Product Name	beta-Amyloid (1-42)
Chinese Name	β 淀粉样肽（1-42）抗体
Alias	beta-Amyloid(1-42); A4; AAA; ABETA; ABPP; AD1; Alzheimers Disease Amyloid Protein; Amyloid B; Amyloid Beta A4 Protein Precursor; Amyloid Beta; Amyloid of Aging and Alzheimer Disease; APP; APPI; B Amyloid; Beta APP; Cerebral Vascular Amyloid Peptide; CTFgamma; CVAP; PN II; PN2; PreA4; Protease nexin II; A beta; Amyloid 1-42.
Research Area	Cell biology immunology Neurobiology Alzheimer's
Immunogen Species	Mouse
Clonality	Polyclonal
React Species	Human(predicted:Mouse,Rat,Chicken,Dog,Pig,Cow,Rabbit)
Applications	WB=1:500-2000 （Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Theoretical molecular weight	4.4kDa
Cellular localization	The nucleus cytoplasmic The cell membrane Extracellular matrix
Form	Liquid
Concentration	1mg/ml
immunogen	KLH conjugated synthetic peptide derived from human beta-Amyloid: 1-42/42 <Cytoplasmic>
Lsotype	IgG
Purification	affinity purified by Protein A
Buffer Solution	1M TBS(pH7.4) with 1% BSA, 3% Proclin300 and 50% Glycerol.
Storage	Shipped at 4℃. Store at -20 ℃ for one year. Avoid repeated freeze/thaw cycles.
Attention	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
PubMed	PubMed

Product Detail

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 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. 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 brain. 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).

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.
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.

SWISS:
P05067

Gene ID:
351

Database links:

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Entrez Gene: 351 Human

Entrez Gene: 11820 Mouse

Entrez Gene: 54226 Rat

Omim: 104760 Human

SwissProt: P05067 Human

SwissProt: P12023 Mouse

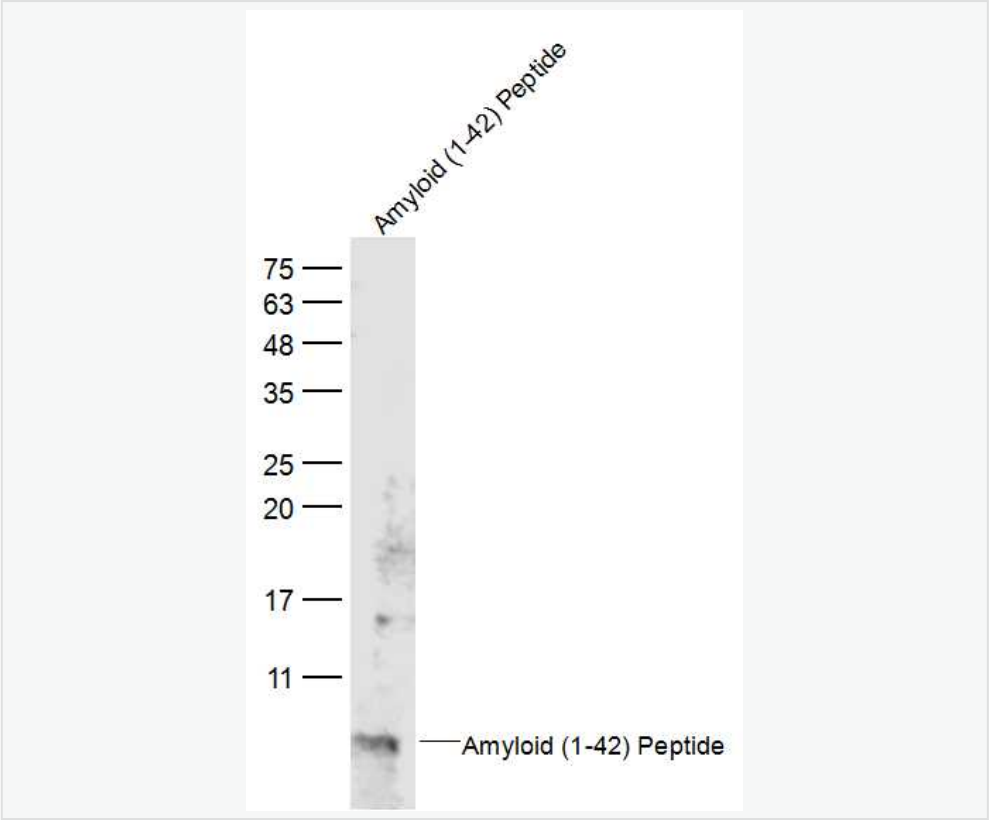
SwissProt: P08592 Rat

Unigene: 434980 Human

Unigene: 277585 Mouse

Unigene: 2104 Rat

Product Picture



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Sample:

Amyloid (1-42) Peptide at 100 ng

Primary: Anti-Amyloid (1-42) Peptide (SL0107M) at 1/300 dilution

Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 4.4 kD

Observed band size: 4.4 kD